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ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

PLEA FOR AN INTERNATIONAL INVESTIGATION INTO OTOSCLEROSIS AND ALLIED FORMS OF DEAFNESS.*

DR. J. S. FRASER, Edinburgh.

I appreciate very highly indeed the honor you have done me in asking me to address you. I hope you will excuse me if, in what I am going to say, I lay special stress on some of the work—unfortunately too meager—which has been done on otosclerosis in Great Britain.

Otosclerosis has been for long the opprobrium of otology. I have nothing startling, or even new, to say about this affection. You will all agree that our knowledge of otosclerosis is in a very vague and unsatisfactory condition. It is for this reason that I venture to suggest the need for an international investigation into the subject. If this can be arranged I believe that it will repay otologists for their time and trouble.

It is a very sad thing to be confronted with a case of deafness, with or without marked tinnitus, in a young or comparatively young woman and to have to confess that in the present state of our knowledge little or nothing can be done to relieve her. I remember Dr.—now Professor—Neumann, telling me that, when he visited the United States, he was sometimes rung up and asked to see a patient in consultation. He asked, "Is the patient a young woman with deafness and tinnitis? If so, I am not coming. I know what the case is already,—otosclerosis."

(*Read before Otological Section, N. Y. Academy of Medicine, May 4, 1923.)
*Accepted for publication in "The Laryngoscope", May 7, 1923.

At the present time otologists spend many hours in making a diagnosis by means of tuning fork and other tests between two kinds of non-suppurative deafness—nerve deafness and otosclerosis—for neither of which they can do much. I often feel that these tests are rather a waste of time.

Everyone will admit that the present position is unsatisfactory. What can be done to alter it?

You will remember that shortly before the war an international investigation was commenced into the subject of ozena. Undoubtedly ozena is a very unpleasant disease which interferes with or destroy the sense of smell. Still hardly anyone will claim for it a place of importance equal to that occupied by otosclerosis. This country is the home of "team work" as applied to the investigation of individual cases. Why should it not take the lead in advocating the creation of an international team to elucidate the subject of otosclerosis?

It may be objected that the present is not a suitable time for such an inquiry. I reply that science is international and that scientists may well set an example.

ETIOLOGY.

(1) *Sex*: Why is otosclerosis so common in the female sex?

On looking over my records of private cases during the last eighteen years (1905-1922) I find that out of 255 cases, 183 were female and only 72 were male patients. From the case records of the Royal Infirmary, Edinburgh, belonging to Dr. Logan Turner and myself, I see that from 1914 to 1922 (inclusive) we had 458 cases of which 354 were female and only 104 male.

(2) *Heredity*: It is stated that a family history of deafness can be obtained in from 30 to 80 per cent of cases—usually about 50 per cent. Gray thinks that the percentage should really be higher than 50, because, as he puts it, "it appears to be an article of faith with some individuals that no qualities are inherited in their own particular families except those which are desirable. We must never accept the patient's statement that there is no deafness in the family." Gray holds that the primary fault is an inherited defect in the organ of hearing and that all other general and local conditions are merely contributory. As regards otosclerosis, Gray looks on the human race as a series in a scale. At one end we find individuals in whom there is almost no potentiality for the development of otosclerosis; at the other end are those in whom the potentiality is exceedingly strong and in whom almost no special stimulus

is necessary to produce the disease. Gray believes that otosclerosis is a variation and that such variations are more apt to occur in organs such as the cochlea which are of comparatively recent origin than in those like the vestibular apparatus which are of more ancient descent. For this reason the margin of the oval window, the footplate of the stapes, and in some cases the nervous structure of the cochlea are affected while the other parts of the labyrinth are untouched. Yearsley believes that otosclerosis is transmitted in the female line, but this is doubtful. Koerner has suggested that otosclerotics should not marry but this seems to me rather a "large order."

(3) *Congenital or Developmental Deafness and Otosclerosis*: Cases of congenital deafmutism have been examined by Politzer, Haberman, Schwabach, Panse, Denker, Alexander, Lindt and Nager, in which a focus of otosclerosis was found in the labyrinth capsule. The connection, if any, between the two conditions is by no means clear. Alexander believes that there is a stepping-stone between congenital deafness and the various forms of congenital hardness of hearing, that the first rudiments of otosclerosis are often of congenital origin and that the ankylosis of the stapes and atrophy of the sensory epithelium are secondary. These congenital foci may exist without giving rise to symptoms until the age of puberty. Koerner believes that otosclerosis is due to certain determinants in the germinal cells of the parents, but is influenced by puberty, parturition, the climacteric period, and also by disease of the middle ear. He explains cases of otosclerosis with no apparent heredity by supposing that the disease has skipped some generations. Hagener believes that the primary condition in otosclerosis is a change in the eighth nerve, and, in support of this, points to cases in which tinnitus is the first symptom of the disease. Manasse has shown that the nerve changes are the same in advanced otosclerosis and in pure nerve deafness. We know that there are cases of atypical otosclerosis in which functional examination apparently shows pure nerve deafness, and yet, on microscopic examination, a typical focus is found in the labyrinth capsule. Such cases have been recorded by Alexander, Kalenda, and Stern. This form of the disease is markedly hereditary. Manasse finds that in otosclerosis there is little or no connection between the changes in the labyrinth capsule and the results of clinical examination. Even with a small bony focus we may have the clinical signs of otosclerosis, and, on microscopic examination, the well-marked picture of nerve deafness. Indeed, in one case there was, in the better hearing ear, a focus of otosclerosis,

while on the other side the labyrinth capsule was normal. Goerke apparently believes that the focus of pathological bone is only incidental and *not* of great importance. Nager, however, holds that, if the otosclerosic focus adjoins the stapedio-vestibular ligament, we have the well-known Bezold's triad of signs.

Stein is of opinion that in otosclerotics there are always other diseases of degenerative origin. Gray regards otosclerosis as a degenerative process and suggests still another name for the disease—"idiopathic degenerative deafness." Hammerschlag notes that the bony changes characteristic of otosclerosis have been found in many cases of congenital deafness—not only in the hereditary degenerative (sporadic) type but also in endemic cases. He reminds us that there are families in which cases both of hereditary deafness and otosclerosis are to be found. Indeed he regards otosclerosis, progressive nerve deafness and hereditary deafmutism as different manifestations of the same hereditary disease.

Edinger has suggested that hereditary deafness appearing in middle age belongs to the group of wasting diseases, in which the normal demands of the organ cannot be replaced. Progressive nerve deafness is obviously to be included in this group and Goerke wishes to put otosclerosis into the same category.

(4) *Age*: The age incidence of otosclerosis is difficult to estimate. I find it very common in girls and young women between 18 and 26 years. Probably slight deafness has been present for some years before the patients seek advice so that it is likely that the real onset of deafness occurs about the age of puberty.

(5) I need not enumerate all the various toxic and other conditions to which otosclerosis has been attributed. I may, however, mention tonsillar, dental and intestinal sepsis, pregnancy and the puerperium. For once syphilis does not seem to be a guilty party. The results of the Wassermann reaction and the greater frequency in the female sex are against syphilis.

(6) A dystrophic change in the nerve supply of the region is said by some to be the primary factor in the production of otosclerosis. We know that Froeschels has described the tickling symptom as absent or greatly reduced in cases of otosclerosis but the value of this finding has been discounted since it was also reported in other ear diseases. Further it is also possible that the dystrophy may be due to the presence of the lesion.

(7) Abnormalities of the endocrine glands have been advanced as the main factor in the production of otosclerosis. No doubt one

appears very "up-to-date" nowadays when one attributes an obscure disorder, such as otosclerosis, to an affection of the ductless glands, but what proof have we of the connection, if any, between the two? The ductless glands are "all the rage" at present. It is true that changes in the labyrinth capsules similar to those seen in otosclerosis have occasionally been found in cretins and in patients suffering from goitre. In most cases, however, of endemic deafness and deaf-mutism no such changes were discovered. Italian otologists tell us that otosclerosis is common in cases of osteomalacia—a condition in which the ovaries and suprarenals are said to be involved. Recently work has been done which tends to show that in otosclerosis the calcium content of the blood is diminished. Lastly disorders of the pituitary and parathyroid glands are credited with producing pathological changes in the bony skeleton. I certainly have never seen any improvement in otosclerosis from the administration of extracts of the ductless glands, singly or in combination. The subject calls for investigation, but I am afraid that at present the use of the words "disorder of the endocrine glands" is regarded by many as rather comforting—like that of the blessed word "Mesopotamia."

(8) *Past Attacks of Otitis Media*: On this point opinions differ greatly and it is very difficult to clear up the question. Almost all children have had at one time or another an attack or attacks of earache, so that it is impossible to exclude otitis media as a cause of otosclerosis. The usual objection is that the drumheads are often normal but does this exclude the possibility that such cases may be secondary to otitis media? I do not think so. Is it not possible that, while the membrana tympani returns to normal, the local infective process may linger in the region of the anterior margin of the oval window—a *locus minoris resistentiae?* On the other hand even the supporters of the "otitis media" view—among whom I place myself—do not claim that inflammation of the middle ear explains everything. (All cases of purulent rhinitis due to scarlet fever or measles do not end in ozoena, nor does every case of bronchitis end in bronchiectasis.) In the vast majority of cases otitis media passes off and leaves no deafness behind nor any changes in the drumhead. This I have observed in my own children. Can we be quite sure, however, that in patients with an hereditary tendency to otosclerosis the inflammatory condition may not in time invade the bone? In certain families the auditory apparatus may be congenitally weak and therefore unable to throw off completely an attack of otitis media. Bryant states that he believes in the con-

nection between naso-pharyngeal catarrh and otosclerosis. Lucae in his book on chronic progressive deafness stated that no sharp dividing line could be drawn between chronic middle ear catarrh and otosclerosis.

Nowadays it is usual to divide cases of otosclerosis into Primary and Secondary, the latter being those which result from otitis media. Professor Gustav Alexander put it to me thus: "We know that some cases of otosclerosis follow otitis media; the question is—do all cases?"

(9) *Congenital Weaknes of the Mesoblastic Tissue*: The conjunction of otosclerosis with Fragilitas Ossium and blue sclerotics is of considerable interest. I have examined three such cases all from one family. All three structures—the labyrinth capsule, the long bones and the sclerotics—arise from the mesenchyme.

(10) *Primary Affection of the Labyrinth Capsule*: Many otologists look on otosclerosis as a disease which occurs primarily in the bone of the labyrinth capsule, the infection coming by way of the blood stream as in osteomalacia, rheumatism, syphilis, etc. We must remember, however, that formerly tonsillitis was held to be of rheumatic origin, i. e., that the tonsil was supposed to be infected through the blood stream. Now we believe that many cases of rheumatism are of tonsillar origin, i. e., that the blood stream is infected from the tonsil. In the same way in my opinion the number of cases of otosclerosis which are regarded as secondary to otitis media is likely in the future to grow larger while those looked upon as "primary" will probably decrease.

There is too great a tendency to attribute otosclerosis to one cause alone. Is it not possible that several causes may be combined? I hold that an attack of otitis media may be compared to the "match" or "cigarette end" that lights the fire. The hereditary tendency and the female sex correspond to the "inflammable material." Any weakening of nerve influences and disorders of the ductless glands which preside over the processees of bone formation and repair may be likened to "a want of water with which to extinguish the fire."

PATHOLOGY.

The trouble in regard to the pathology of otosclerosis is that few general pathologists appear to know much about the diseases of bone and that few otologists can be regarded as expert pathologists. The labyrinth and its capsule are not easy tissues to prepare for microscopic examination. I had long wanted to obtain the opinions of eminent pathologists regarding the nature of the changes seen

in otosclerosis when, in 1919, there was a meeting in Edinburgh of the Pathological Society of Great Britain. Though not a member, I was asked to contribute a paper and I thought it would be a good opportunity to collect opinions. In order that the assembled professors and others might hear and see both sides of the question, I asked Dr. Gray of Glasgow to attend so that we both might put our views before the meeting. Well, we showed our microscopic preparations, gave our own opinion of their interpretation and waited for the words of wisdom. The assembled pathologists showed

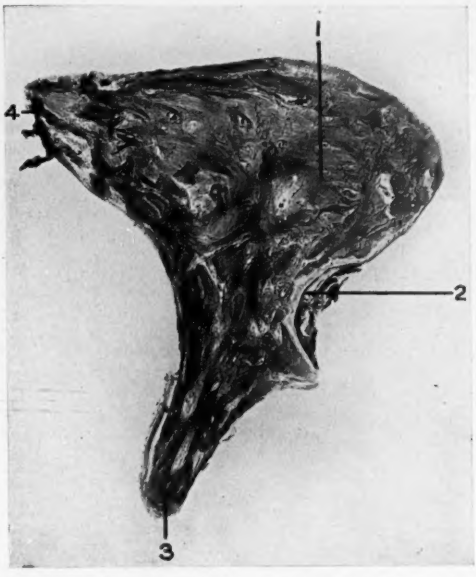

Fig. 1. Vertical transverse section of normal incus. 1, body of incus; 2, articular surface (with blood clot); 3, long process; 4, short process. (x 15.)

a surprising unanimity; they said nothing. I got up again and explained that the matter was a very important one as the disease affected young and often very pretty girls. Even that failed to elicit any opinion as to the pathological nature of the affection.

We have only to remember the number of names given to the condition commonly known as otosclerosis to realize that there is no general agreement as to the pathology. I need not enumerate these titles for they are well known to all of you.

Observers admit that the first stage is a spongification of the bone with the formation of large spaces containing a central blood vessel surrounded by granulation or connective tissue. The bone forming the walls of this space stains deeply with basic stains such as haematoxylin. Some believed that this is new formed bone, but in my experience, new bone takes on the eosin stain. I believe that the deeply staining bone is merely the old bone tissue, the staining reaction of which has been altered by the change it has undergone owing to the vascular dilatation and increased supply of inflammatory lymph.

Secondly observers admit that the final stage is one of sclerosis or formation of dense more or less irregular lamellar bone which

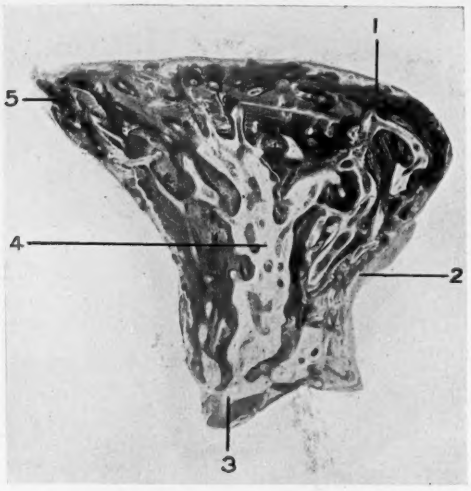

Fig. 2. Eroded incus from a case of chronic middle ear suppuration. 1, body of incus; 2, articular surface; 3, eroded long process; 4, invasion of the marrow spaces by a chronic form of osteomyelitis analogous to that seen in the labyrinth capsule in otosclerosis and congenital syphilitic deafness; 5, short process of incus. Note large marrow space with dilated vessels extending from region of eroded long process. (x 15.)

stains with eosin. Differences of opinion are revealed when we come to the presence of osteoclasts. Some hold that these cells are responsible for the removal of bone in the early stages of the disease, while others—probably the majority—hold that osteoclasts only appear at a more advanced stage. In the early period bone absorption is probably due to pressure of the new formed granulation tissue. In any case osteoclasts are not necessary for bone absorption as one sees from the examination of the labyrinth capsule

in cases of tumor of the eighth nerve where the internal meatus is greatly dilated and yet no osteoclasts can be found.

Undoubtedly the process of bone absorption and the formation of large vascular spaces appears to proceed along the blood vessels which can be seen to enter the labyrinth capsule from the mucoperiosteum in the anterior margin of the oval windows.

Bony ankylosis of the stapes is probably a late feature of the disease. It was present in only two of Gray's four cases and both of these were of many year's duration. In my early case it was not present but in the second case, an old woman of 65 years, it was marked on both sides.

Otosclerosis is not always confined to the site of election. The region of the bony prominences of the lateral canal may be involved as in my second case. Further the affection may extend round the cochlear capsule between the lamellar and the cartilage bone to the region of the internal meatus. Politzer held that the presence of otosclerosis in this latter situation proved that the disease was not due to otitis media as there was no mucosa present here. There is, however, a lymph or connective tissue space between the cartilage and lamellar bone—well marked in the foetus and in young children, less evident in adult life, along which the chronic inflammatory process may spread.

As Katz has pointed out a change may occur in the ossicles in cases of otosclerosis similar to that seen in the labyrinth capsule. I have observed much the same thing in the malleus and incus in cases of chronic middle ear suppuration and cholesteatoma. (Fig. 1 and 2) We must remember that the tympanic ossicles and joints are almost the only bones and joints in the body which are covered merely by mucous membrane and so liable to infection from the surface. We know how often inflammatory affections of the nose lead to bone disease of the turbinals, e. g., cases of nasal polypus and ethmoiditis.

The anterior margin of the oval window is a weak spot. This may be due to the presence of the tensor tympani tendon or to the greater movement of the anterior part of the foot plate of the stapes, as Bruhl suggests, but it appears more probable that its vulnerability is due to the entrance of vessels from the deep layer of the mucosa, associated with the presence here of fibrous tissue strands which replace cartilaginous "rests" well seen in the foetus and young subjects. (Fig. 3) This "rest" of cartilage is connected by a narrow strip to the anterior margin of the oval window. I have observed that this strip divides like a Y—one end becoming continu-

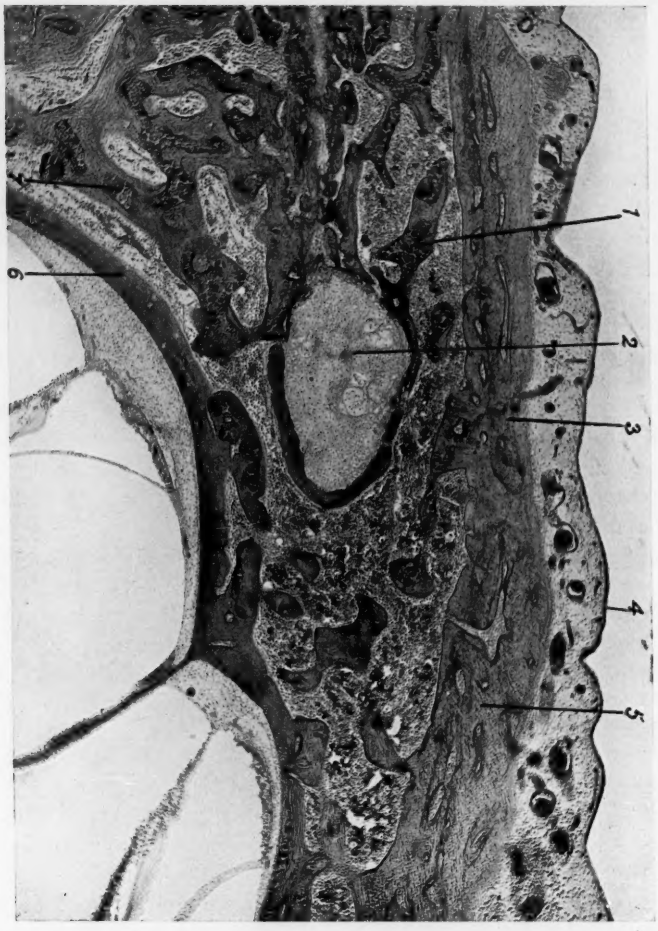

Fig. 3. Anterior margin of oval window from full term foetus showing remains of foetal cartilage. 1, cartilage or interglobular bone; 2, rest of foetal cartilage; 3, lamellar or periosteal bone; 4, mucosa of tympanum; 5, lamellar or periosteal bone; 6, endosteal bone; 7, cartilage or interglobular bone. (x 45.)

ous with the endosteum of the labyrinth anterior to the stapes foot plate, while the other joins the mucoperiosteum of the window niche. Mayer states that the island of cartilage may remain unossified until after the second year—indeed ossification may never occur and a medullary space may remain filled with fatty masses. Mayer points out that two centres of ossification meet at the site of election—one from above and the other from below. These findings lend support to the view of Neumann and others that otosclerosis is a congenital anomaly of the process of growth in the petrous pyramid —an anomaly which only gives rise to symptoms (deafness and tinnitus) after the age of puberty. The idea is that, while the growth of the petrous bone should normally cease in childhood, in otosclerotics the growth goes on and results in the formation of vascular spongy bone.

Mayer thinks that otosclerosis may be in the nature of tumor formation (hamartoma). In two or three cases he found that otosclerosis was associated with the presence of a small neurofibroma of the eighth nerve and thinks this fact significant. Many years before, Gray had likened the area of otosclerosis to an enucleable foreign body in the labyrinth capsule and had suggested that it might be a sort of a "aseptic necrosis" due to interference with the blood supply. Mayer also attributes importance to these circulatory changes which may lead to reconstruction of the bone in the affected area.

Bryant has called attention to the similarity of the changes seen in otosclerosis to those observed in rickets, osteomyelitis, ostitis fibrosa (v. Recklinghausen), osteomalacia osteitis deformans (Paget), osteoarthritis and syphilitic bone disease. Deafness, tinnitus and giddiness occur in cases of ostitis fibrosa, while on microscopic examination exostoses and stapes ankylosis have been found. Mayer, Nager and Jenkins have all called attention to the change in the labyrinth capsule in the case of osteitis deformans. Siebenmann has compared spongification of the labyrinth capsule to the change seen in a rib in empyema.

The question arises: Is otosclerosis a pathological entity? Are we examining different stages of the same disease or are we observing different diseases?

Case 1. Male, aged 19, had suffered from chronic middle ear suppuration of unknown origin for many years. At the autopsy a large temporo-sphenoidal abscess was discovered. A vertical saw cut was made through the temporal bone from the external ear to the internal meatus in order to demonstrate cholesteatoma in the tympanic cavity and attic. Later it was determined to examine the ear

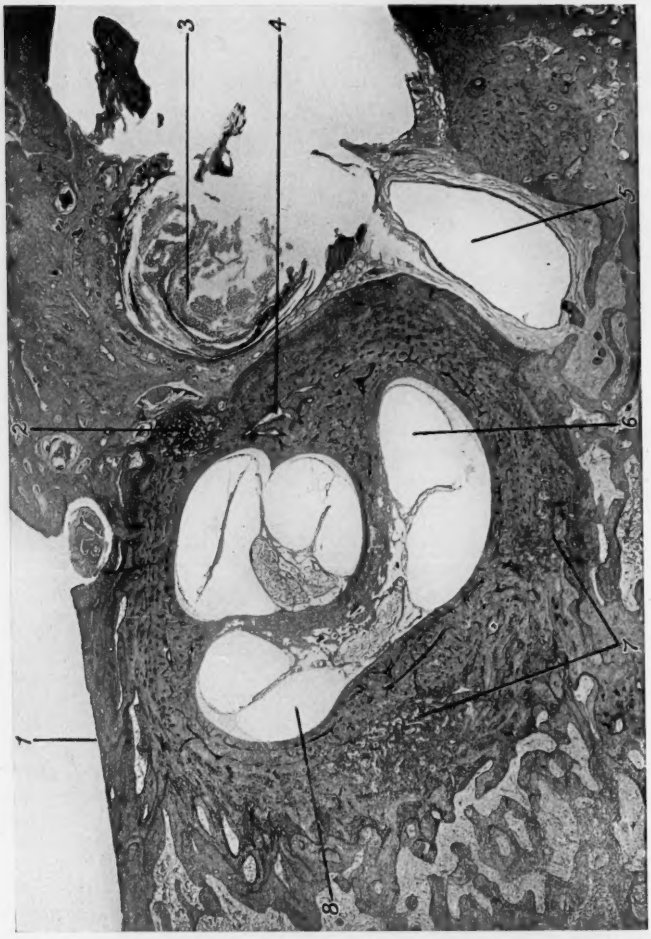

Fig. 4. Vertical transverse section through cochlea, showing three areas of ostitis vasculosa (otosclerosis). 1, middle fossa; 2, area of ostitis vasculosa; 3, cholesteatoma; 4, remnant of lymph space in capsule of cochlea; 5, tubal part of tympanic cavity—the tympanic cavity in this case is divided into an upper part (lined by cholesteatoma) and a lower part communicating with the Eustachian tube. 6, scala vestibuli of basin coil; two areas of ostitis vasculosa; 8, scala tympani of basal coil. (x 8.)

microscopically. The tympanic cavity, aditus and antrum were lined by cholesteatoma. On the inner wall of the tympanum, in the anterior margin of the oval window, there is an area of ostitis vasculosa in the labyrinth capsule. We can trace the spread of the disease in the wall of the labyrinth between the periosteal and cartilage bone round to the region of the internal auditory meatus, and also over the apex of the cochlea to the anterior part of the bony capsule of this structure. At the apical coil of the cochlea the spongification reaches the perilymph space. (Fig. 4)

In order to explain how the chronic infective condition, which invades the labyrinth capsule from the region of the promontory, can reach the internal auditory meatus it is necessary to recall a few facts regarding the development of the ear. About the third month of foetal life we find the membranous labyrinth surrounded by a capsule of cartilage. Later this cartilage capsule is enclosed in lamellar bone formed by the deep layer of the tympanic mucosa and the osteogenetic layer of the dura matter. A lymph space however exists between the cartilage bone and the lamellar bone for some time after birth. Later only traces of this space can be seen (Fig. 6), but there is always a "potential" space as between two pieces of wood which have been glued together. If a chronic inflammatory process involves the labyrinth capsule and opens up this space, it may spread round the cochlea and reach the region of the interior meatus.

Case 2. Female, aged 63 The patient attributed her deafness to an attack of scarlet fever and otorrhea in childhood. She complained of severe tinnitus like the roaring of the sea. The noises also had at times a "beating" character. *She sometimes complained of dizziness. She heard better in a noisy place.*

Microscopical examination of right ear. The drumhead is slightly *thickened* in the posterior part. *In the region of the oval window the submucosa is thickened, congested and oedematous. The stapes is ankylosed* by bone to the anterior margin of the oval window. *Above the* level of the *oval window there is an area of* osteoporosis or *ostitis vasculosa. Engorged vessels are seen entering this area from the deep layer of the swollen tympanic mucosa.* The ostitis vasculosa shows a tendency to extend forward into the cochlear capsule between the cartilage and lamellar bone. (Fig. 7 and 8.)

Left ear. Otitis media (purulenta) is still present and the *drumhead is perforated.* There is a *marked chronic adhesive process in the upper part of the tympanum,* with great thickening of the sub-

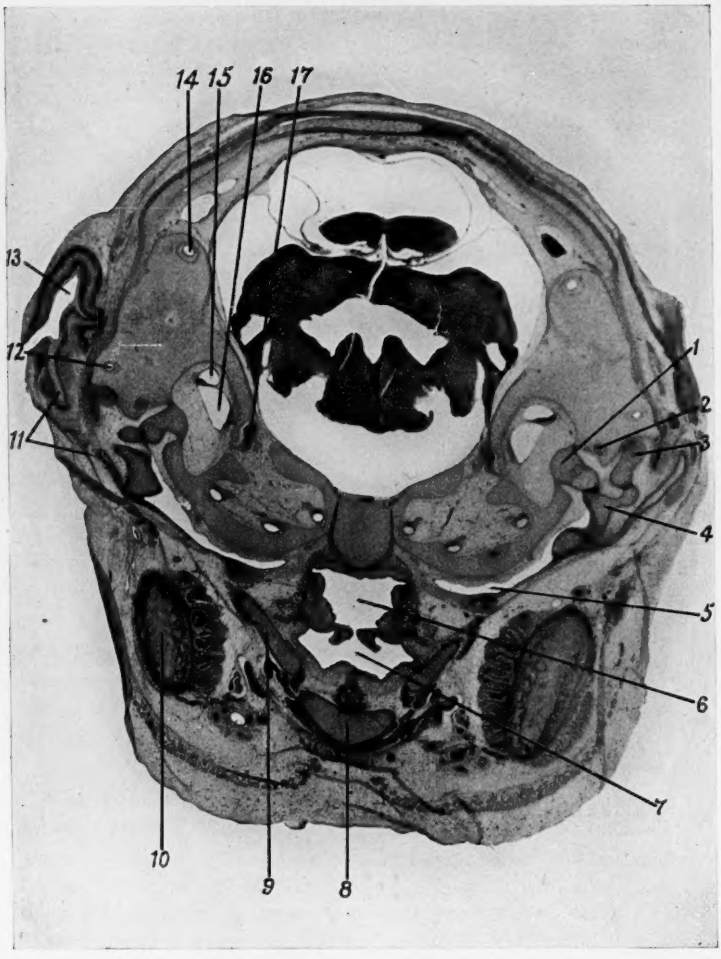

Fig. 5. Vertical transverse section through head of foetal pig showing the foetal cartilaginous capsule of the labyrinth on both sides. The endolymph spaces are patent, but the perilymph spaces are still filled with foetal connective tissue. 1, footplate of stapes; 2, facial nerve; 3, incus; 4, malleus; 5, Eustachian tube; 6, naso-pharynx; 7, mouth cavity; 8, hyoid; 9, Meckel's cartilage; 10, lower jaw; 11, external auditory meatus; 12, external canal; 13, hollow of auricle; 14, superior canal; 15, utricle; 16, saccule; 17, cochlea nerve. Note the cartilage capsule of the labyrinth extending from above 14 to the basi sphenoid which lies above 6. (x 8.)

mucosa and erosion of the long process of the incus. *The stapes is ankylosed* to the anterior margin of the oval window. There are three well marked areas of *otitis vasculosa* in the labyrinth capsule *on the inner wall of the middle ear;* (a) In *the lateral canal;* (b) in the *anterior margin of the oval window;* (c) at the *apex of the cochlea.* The labyrinth contents are normal. (Fig. 9-12.)

In this case the otosclerosis on both sides was secondary to otitis media. On the right side the otitis media passed off but left traces in the region of the tube and oval windows. In time the inflammatory process invaded the bone in the latter area and resulted in otosclerosis with fixation of the stapes. On the left side the otitis media continued and invaded the labyrinth capsule in three distinct areas as stated above.

Case 3. Male, aged 27, had chronic middle-ear suppuration on the left side for ten years. Ten days before admission the ear ceased to discharge, and three days later the patient began to have occipital and frontal headache. For one week there had been *facial paralysis on the left side. Four days before admission vomiting* commenced and has continued. Of late patient has complained of *giddiness on getting out of bed, and has tended to fall to the left side.*

Examination. The right drumhead shows a large retracted scar. The *left external meatus contains foetid pus and granulations.* Cochlear apparatus: *Schwabach lengthened; Weber laterialized to right (better) ear examined; Rinne absolutely negative on left side.* On the right side Rinne is *said to be positive!* (This test was *not* carried out by Dr. Turner or the writer.) Patient cannot hear any of the tuning-forks by air conduction on the left side. *With the noise apparatus in the right ear patient is quite deaf. Vestibular apparatus; Patient falls to the left, and the direction of the fall is not altered by changing the position of the head.* Slight *spontaneous nystagmus to the right and slow coarse rotatory nystagmus to the left. Pointing error to the right with both upper extremities. Cold syringing of left ear produced no change* in the spontaneous nystagmus in one and a half minutes. General condition: Temperature *98.4°* F., pulse *88.* Patient lies curled up on the left (diseased) side. Patient is bright mentally. *"Finger nose" test more accurately performed on right side than on left side. Dysdiadokokinesia well marked on left side.* Grasp of both hands good.

Operation on right ear. Track of pus extending from the mastoid surface to the antrum, which was full of cholesteatoma. Fistula from antrum through posterior wall of meatus. Sinus wall normal. *Fistula present in lateral* canal. *Facial nerve* lying *uncovered*

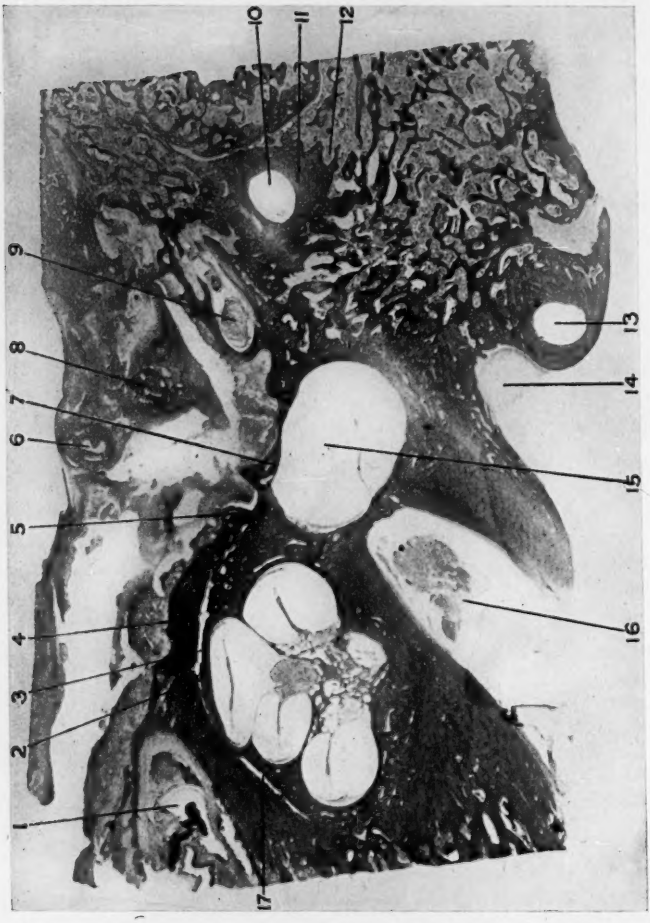

Fig. 6. Horizontal section through right ear of new-born child, showing lymph (3) space between cartilage bone (2) and lamellar bone (4) in the capsule of the cochlea. 1, carotid canal with artery; 2, cartilage bone of cochlea capsule; 3, lymph space; 4, lamellar bone formed from deep layer of tympanic mucosa; 5, anterior margin of oval window; 6, malleus; 7, footplate of stapes; 8, incus; 9, facial nerve; 10, external semicircular canal; 11, cartilage bone surrounding canal; 12, marrow; 13, smooth end of superior canal; 14, fossa subarcuata; 15, vestibule; 16, internal auditory meatus with nerves; 17, lymph space in bony capsule of cochlea. (x 8.)

by bone above oval window. Roof of tympanum and antrum healthy. Dense healthy bone present in triangular area. Posterior canal opened up (*Neumann's operation*); promontory also removed. *Cerebellum explored with negative result.*

Progress. Vomiting present; restlessness; *Kernig's sign* present with *meningitic cry.* Lumbar puncture. *Cerebellum again explored with negative result. Death three days after operation.*

Post-mortem. Basal meningitis. Cerebellar abscess, the size of a walnut, in *anterior part of the left lobe further forward than the exploratory opening* (*although this had been made in front of the sigmoid sinus*). The abscess abuts closely on the fourth ventricle, but no communication is visible. The ventricles of the brain contain slightly turbid fluid.

Microscopical examination of right ear (*chronic adhesive process and otosclerosis*). (Vertical sections from before backward.) There is a large *retracted scar in the drumhead* adherent to the promontory, practically *no tympanic cavity remains.* The attic is filled with delicate connective tissue. The *aditus is obliterated* by new connective tissue. The *stapes* is present, *embedded in granulation tissue but is not ankylosed.* An area of otosclerosis (*osteitis vasculosa*) *is seen just above the basal coil of the cochlea* in the anterior margin of the oval window. (Fig. 13 and 14.) Some giant cells are present in the large vascular spaces of the new bone formation. The *otosclerosis reaches the endosteum of the vestibule* just above the intravestibular part of the cochlea, and *new vascular bone bulges into the vestibule* (endostosis).

This specimen shows that Lucae was right when he said that it was impossible to distinguish between a chronic adhesive process and otosclerosis—i. e., that the one condition ran into the other.

Microscopical examination of left ear. Allowing for the fact that double vestibulotomy had been performed on this side the left ear showed the same changes as those present on the right side. (Fig. 15.)

SYMPTOMS

I will not weary you with a detailed account of the symptoms of otosclerosis which are only too well known. In several cases I have found that tinnitus was present for a considerable period before deafness came on. Further, if nerve deafness is present in a female patient under forty years and if the Wassermann reaction of the blood and cerebrospinal fluid are negative, I am very suspicious that the case is one of atypical otosclerosis, especially if there is a history of deafness in the family. Many of my cases of otosclerosis laid stress on the fact that they were

worse during a cold. This is not to be wondered at but one sees it mentioned as a point against the diagnosis of the disease. Deafness may be due to: (1) Stapes ankylosis, but this is a late feature. There may, however, be a certain stiffness of the oval window structures long before ankylosis occurs. This may be compared to the difficulty of sliding out and in the drawers of a badly made bureau. (2) The deafness may be caused by

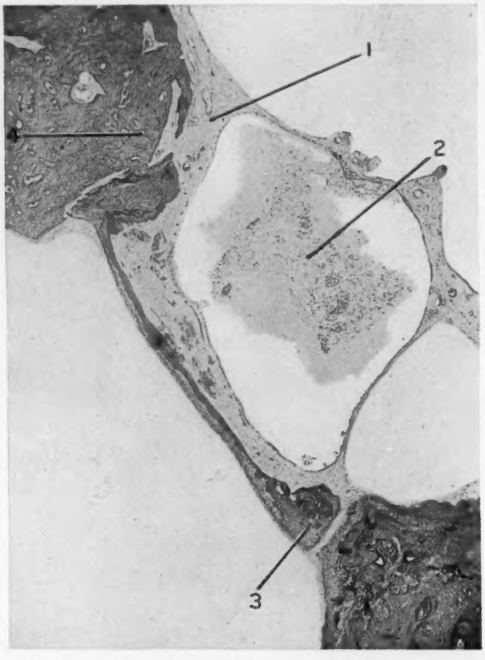

Fig. 7. Otosclerosis associated with otitis media. A. W., female, aged 63. Horizontal section through left ear in region of oval window. No. 210. of anterior margin of oval window; 2, mucopurulent exudate in hollow of stapes; 3, posterior part of footplate; 4, region of ankylosis between stapes and oval window. Note the inflammatory process invading the anterior bony margin of the oval window from the deep layer of the swollen mucosa. (x 25.)

changes in the labyrinthine fluid as suggested by Jenkins and others—a point difficult of settlement. (3) Nerve degeneration may obviously cause hardness of hearing, but this again is usually a late feature. In many cases with marked deafness for speech there are only the tuning fork signs of obstructive deafness while the upper tone limit is normal and the watch is well

heard by bone conduction. (4) I agree with those that hold that the spongification of the labyrinth capsule is the cause of deafness and tinnitus. Normally the nerve endings in the labyrinth are surrounded by dense almost avascular bone and the presence of spongy bone richly supplied with blood vessels in the capsule of the cochlea is bound to interfere with hearing. As so many of the patients say, "I could hear well enough if it were not for the noises."

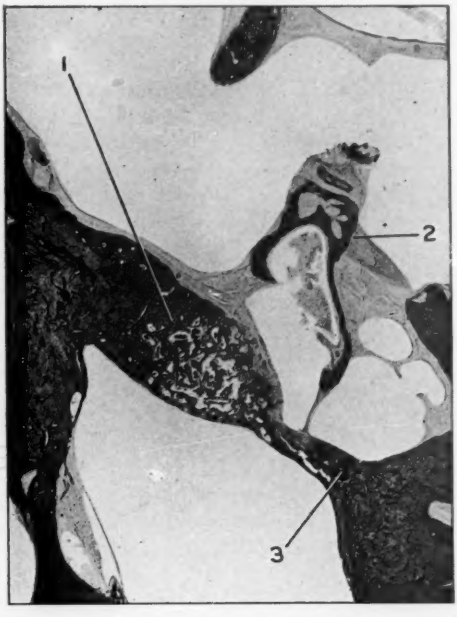

Fig. 8. Otosclerosis associated with otitis media. A. W., female, aged 63. Horizontal section through right ear. No. 265. 1, area of ostitis vasculosa in anterior and lower margin of oval window; 2, tendon of stapedius attached to head of stapes; 3, split in bone of posterior margin of window (artefact). (x 12.)

Late two curious cases were reported by Gray in which the patients heard better during a cold in the head and an attack of hay fever, respectively. Two other cases heard better when a draft of cold air was admitted to an overheated room. The second of these patients took a little brandy and then suddenly experienced a great improvement in hearing. I have been told of a case in which an old deaf man suddenly heard very well

just before death. It is said that the inhalation of amyl nitrite or chloroform, or the administration of pilocarpin or even a warm douche to the ear may cause sudden improvement in hearing. The explanation of these phenomenon may be either an alteration of the labyrinthine tension or of the local circulation.

Paracousis: Politzer believed that paracousis was due to the vibration produced by the noise of machinery, for example, exercising a sort of "massage effect" on the ossicles and joints. On

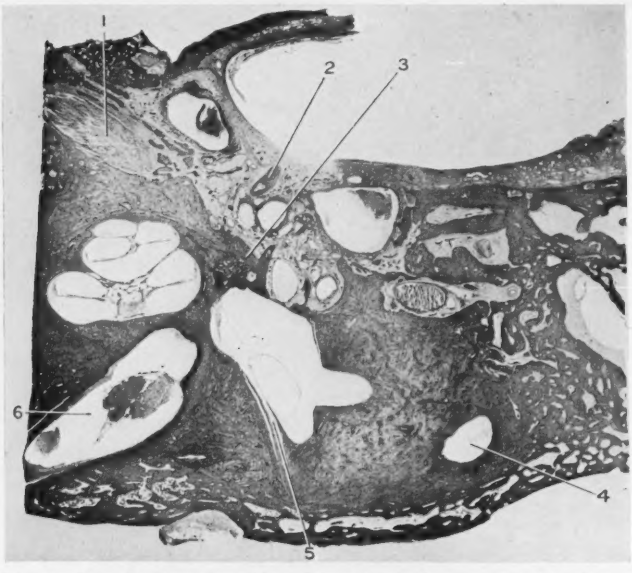

Fig. 9. Otosclerosis associated with otitis media. A. W., female, aged 63. Horizontal section through left ear in region of oval window. No. 210. 1, tensor tympani; 2, malleus handles; 3, area of ostitis vasculosa (otosclerosis) in anterior margin of oval window, with anterior part of stapes ankylosed to it; 4, posterior canal; 5, endolymphatic duct; 6, internal meatus. Note the marked adhesive process in the tympanic cavity. (x 6.)

this view one would expect that the beneficial action would continue for some time after the noise had ceased, but, as a matter of fact, it does not do so. Treatment based on Politzer's assumption did no good, e.g., inflation, the pressure probe and vibratory massage. Fibrolysin was also used on this theory, but without any result. A more reasonable view is that according to which the extraneous noises are necessary to stimulate the atrophic nerve apparatus.

Jenkins and others hold that paracousis is more apparent than real, that it is due to the fact that extraneous noises like those in a train or street car, which are low in pitch, do not disturb an otosclerotic so much as a normal hearing person and that therefore the otosclerotic gets more advantage from the raising of his friend's voice which always occurs under such conditions. Jenkins proved this by getting someone to read to a normal per-

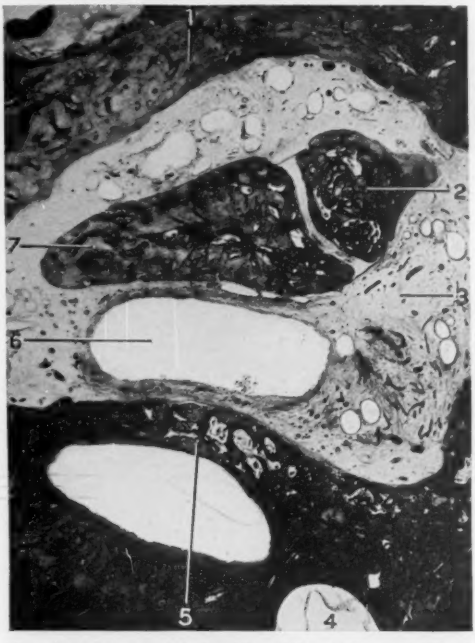

Fig. 10. Otosclerosis associated with otitis media. A. W., female, aged 63. Horizontal section through left ear in region of attic. No. 65. 1, external wall of attic; 2, malleus; 3, swollen submucosa; 4, ampullary end of superior canal; 5, area of ostitis vasculosa in bony wall of lateral canal; 6, remains of attic cavity; 7, short process of incus. (x 12.)

son and an otosclerotic in a quiet room in which a noise was suddenly produced. The normal person did not notice that the reader's voice had been raised at all when the noise occurred, but the otosclerotic found it very marked.

DIAGNOSIS.

Can we be sure that a given case is one of otosclerosis? Certainly, if we have a family history of deafness, normal drum-

heads, flamingo tinge of the promontory, Bezold symptom triad, normal Eustachian tubes and no improvement on inflation.

I have shown you, however, three cases in which otosclerosis was combined with middle ear suppuration or with an adhesive process. How can we then diagnose otosclerosis? It is not so easy, but if we have marked or almost complete deafness, severe tinnitus, great elevation of the lower tone limit, and paracousis, it is at least extremely probable that secondary otosclerosis is present.

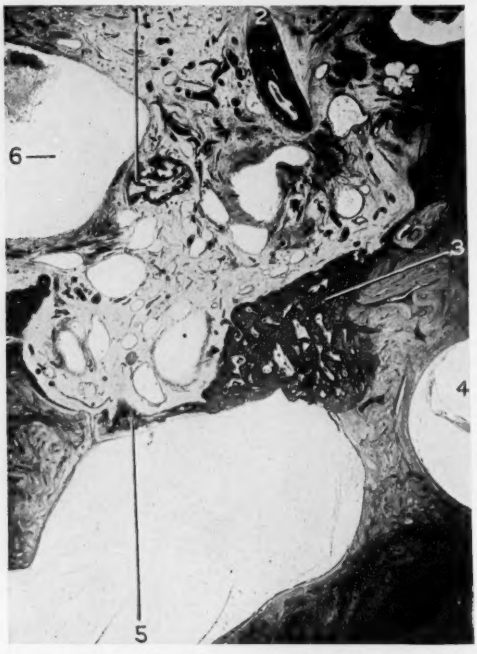

Fig. 11. Otosclerosis associated with otitis media. A. W., female, aged 63. Horizontal section through left oval window. No. 205. 1, long process of incus, showing ostitis vasculosa; 2, malleus; 3, area of ostitis vasculosa in promontory ankylosed to 5, footplate of stapes; 4, cochlea; 6, tympanic cavity. (x 12.)

Jenkins points out that in middle ear deafness apart from otosclerosis, Rinné's test only becomes negative when the hearing distance is considerably reduced, e.g., twelve feet for the conversation voice. In otosclerosis on the other hand Rinne's test is negative even though the patient can hear the conversation voice at six feet. He also calls attention to the confident affirmative answer of

the otosclerotic when asked if she can hear the fork on the mastoid after she has ceased to hear it by air conduction. This contrasts with the hesitating answer of the patient who has only an adhesive process in the typanum. The same remarks apply to the lateralization of the Schwabach test.

TREATMENT

In Edinburgh I am sorry to say that, since we have learned to diagnose otosclerosis, we have not been very enterprising in

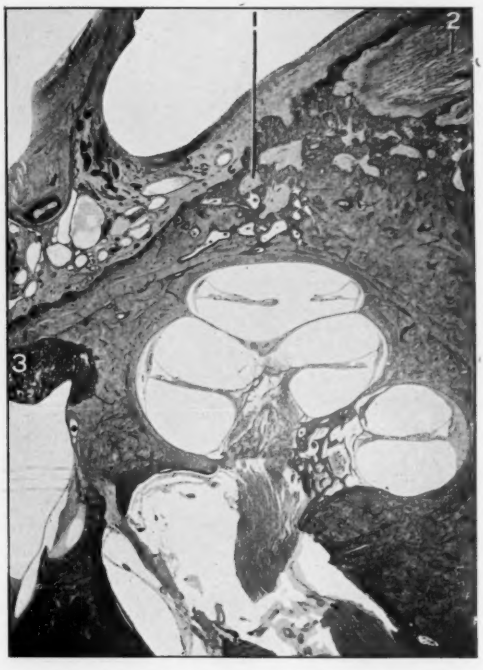

Fig. 12. Otosclerosis associated with otitis media. A. W., female, aged 63. Horizontal section through left cochlea. No. 245. 1, area of ostitis vasculosa apex of cochlea in region of processus cochleariformis; 2, tensor tympani; 3, area of otosclerosis below oval window. (x 10.)

the treatment of non-suppurative deafness. I have vivid recollections of the weary hours I spent in my first few years of otology in passing the Eustachian catheter and bougie, in massaging the drumhead with Delstanche's apparatus, in injecting fibrolysin and other useless measures. Still, I felt that something should, if possible, be done for these cases and, when I

visited Vienna in 1912, I asked Dr., now Professor, Neumann to give me a "course" on the treatment of non-suppurative deafness by means of galvanism, X-rays, radium, vibratory massage, auditory re-education, etc. Dr. Neumann looked at me and said, "No, Dr. Fraser, I will willingly give you a course on Ohrenschwindel but I will not give you a course on Taschenschwindel." I felt rather snubbed and asked if it were really "as bad as that." I had no wish to become a charlatan.

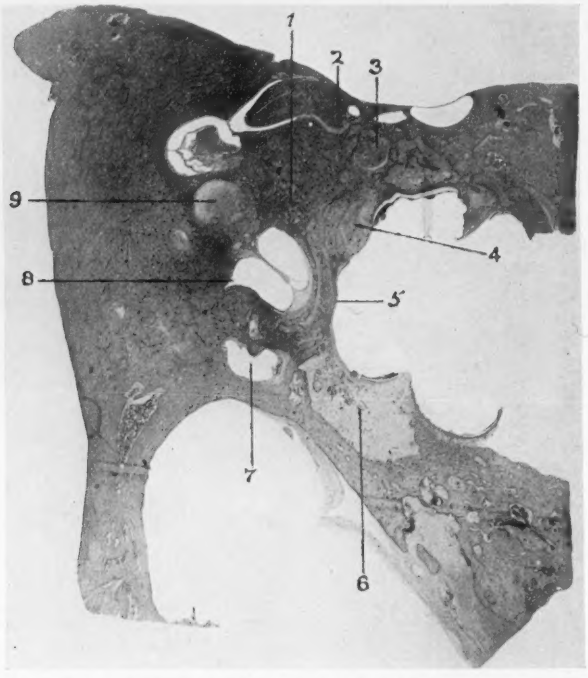

Fig. 13. W. F. Section through right ear, No. 220, x 6 diam. 1, area of otosclerosis; 2, geniculate ganglion of facial nerve; 3, tensor tympani; 4, tympanic cavity obliterated; 5, promontory covered by squamous epithelium; 6, hypo-tympanic cavity obliterated by connective tissue; 7, niche of round window; 8, cochlear opening of perilymphatic aqueduct; 9, anterior wall of sacculus.

I do not know if this accurately represents the present state of affairs but, if so, are we going to take it "lying down?" We may be willing to explain to an old man of 70 or 80 years that deafness is frequently produced by changes which occur in the inner ear as the result of anno domini, i.e., of arteriosclero-

sis. We may be prepared to tell a patient with congenital or acquired syphilitic deafness that little or nothing can be done, for, in my experience salvarsan and its substitutes have no effect; mercurial inunctions and iodides are more hopeful. Are we, however, to be content with saying to a young girl or a

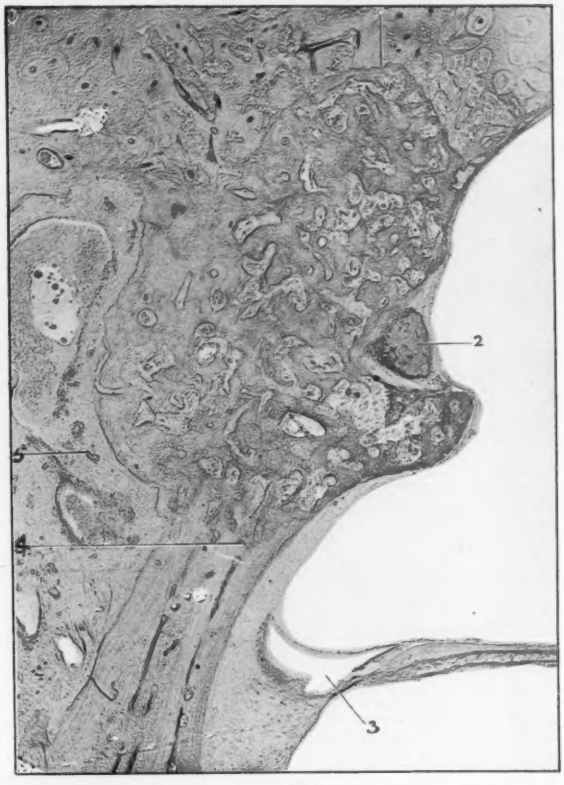

Fig. 14. W. F. Vertical section of right ear, No. 255, x 35 diam. 1, upper edge of otosclerotic area; 2, footplate of stapes not ankylosed; 3, intravestibular portion of scala media; 4, lower margin of otosclerotic area; 5, connective tissue filling niche of oval window.

young man that the case is one of chronic and unusually progressive deafness for which, in the present state of our knowledge, very little can be done, and to suggest lip-reading if the patient is young enough to take it up and is the kind of person who "looks you in the face?" If not, we can advise an arti-

ficial aid to hearing and write a short and unsatisfactory account to the patient's doctor in regard to the treatment of tinnitus. I hope to hear this evening that others are more successful.

Gray points out that there is no routine treatment for otosclerosis; every case must be a study in itself. He admits that the results of treatment, so far as improvement goes, are generally unsatisfactory. In a large number, especially in the very early stages, the disease may be arrested or greatly retarded. In a few some improvement may be obtained. If the affection comes on early in life the outlook is extremely bad. Cases with hereditary otosclerosis are unfavorable; if tinnitus is marked,

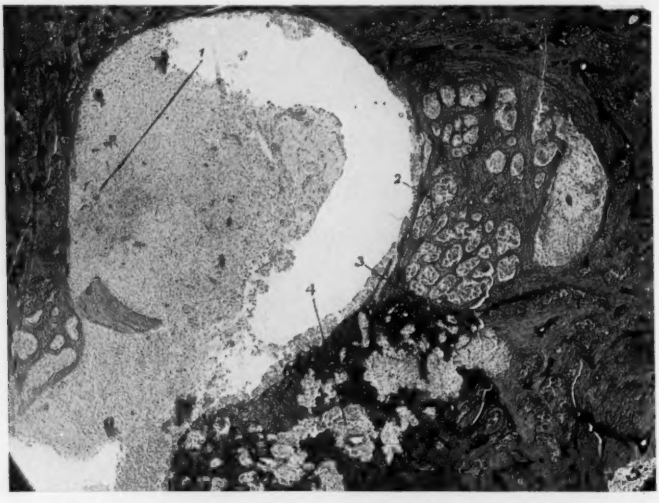

Fig. 15. W. F. Vertical section of left ear, No. 160, x 35 diam. 1, purulent exudate in vestibule; 2, terminal branch of vestibular nerve; 3, upper end of otosclerotic area; 4, vascular space of otosclerotic area showing recent acute inflammatory infiltration.

the case is likely to progress rapidly. In the pure form of the disease, unassociated with catarrh of the pharynx or middle ear, a moist climate has no particularly bad effect, but severe cold is unfavorable. Gray lays great stress upon diet, and states that meat, which permits of considerable toxin formation, has a bad effect. Alcohol, tobacco, strong coffee and tea are also unfavorable. The injurious effect of pregnancy and the puerperium have been rather over-estimated. Dieting, laxatives, intestinal antiseptics, the elimination of septic foci and vaccine

treatment all have a place. We can, according to Gray, do something better than merely tell the patient to learn lip-reading or to use an artificial aid to hearing. The value of the former has been considerably exaggerated. z

Sir Robert Woods injects a preparation of iodine into the tympanic cavity with good results.

About twelve years ago Charles Heath of London, England, published a paper in which he described his method of treating cases of deafness associated with paracousis—cases which he described as those with "loose ear drums," or "pocket handkerchief deafness." Heath believed that the pathology of this affection consisted in relaxation of the drumhead from too frequent and forcible inflations of the middle ear—inflations which caused the membrane to lose its proper tension, the tendons of the tympanic muscles to become relaxed and the labyrinthine tension to be unduly lowered. You will remember that Heath's method consisted in painting the drumhead at frequent intervals with increasing strengths of blistering fluid. At the urgent request of a friend of my own, a medical man suffering from otosclerosis secondary to otitis media, I tried the method, but without any beneficial result. Gray treated twelve cases according to Heath's method but in not a single instance was there any improvement.

The method of auditory re-education associated with the names of Zund-Burguet and Maurice has also given unsatisfactory results according to the reports of Gray and of Muecke. My own experience is limited as I did not carry out this treatment myself. Several of my patients, however, were treated by the late Major Porter but without benefit as far as my observation went.

Lately I saw a patient who told me that she had been diagnosed ten years ago as a case of unilateral otosclerosis. She had been informed that she would gradually become deaf, first of all in the already affected ear and later on in the sound one, in all probability. I examined the patient and confirmed the previous diagnosis. I found normal drumheads, lengthened Schwabach, Weber to right (bad ear) and loss of hearing for C32 on the affected side. The voice test, however, showed that the patient could repeat the forced whisper at 12 feet from the affected ear while the left ear was normal for the whisper and watch. I told the patient that I had never known such slow deterioration of hearing in a case of otosclerosis. The patient

then informed me that at intervals during the whole ten years she had been treated by vaccine therapy, the vaccines being made from swabs obtained from the nose and nasopharynx.

Radium: My experience of this line of treatment only amounts to two cases. The first was a girl of 22 years, a typical case, with a history of slight deafness since shortly after puberty, normal drumheads, flamingo-tinge of promontory, etc. Inflation had been tried without benefit. Four applications of radium were given to the right ear, but, though the meatus was protected by tinfoil, the only result was a radium burn of the skin lining the passage. The hearing was not improved. In the other case the result was also unsatisfactory.

X-rays: As far as I know very little work has been done in Edinburgh on the subject. Dr. Hope Fowler tells me that, following the advice of two colleagues in Manchester, he has treated several cases by cross-fire radiations. Some have apparently been benefited while others are stationary. He admits that the period of observation has been too short to draw definite conclusions. Professor Hugo Frey writes to me that he is just about to read a paper on the X-ray treatment of otosclerosis. He seems to be hopeful regarding the method. I should be glad to hear the opinion of those otologists who have treated cases, or have had their patients treated along this line.

Various operations have been performed for the relief or cure of otosclerosis. I will not refer to earlier methods of which I have no experience, but will pass on to th making of a window in the bony wall of the posterior semicircular canal (Barany) or in the lateral or even the superior canal. I have carried out two of these operations at the Royal Infirmary, in each case operating only on the worst hearing ear. In the first case the perilymph space of the lateral canal was probably opened and labyrinthitis resulted. The patient recovered but of course became quite deaf in the operated ear. The second case was more successful. The bony cap of the lateral canal was removed and a skin graft applied. The operated ear is now the better hearing ear, but the improvement was not very great and, though this patient has sent on to me several others suffering from otosclerosis, I have always refused to interfere. It may be that the dissecting microscope used by Professor Holmgren of Stockholm will enable us to operate on the labyrinth with greater accuracy. One feels vaguely that there must be operations

on the ear to correspond with the iridectomy and cataract extraction of the ophthalmologist.

I have had no success with lumbar puncture in the treatment of tinnitus, though Babinski states that 33 per cent of cases were relieved by this procedure.

Our experience in Edinburgh in regard to the destruction of the cochlea in cases of unbearable tinnitus has been very bad. One case of my own, already published, was that of a rather feeble-minded girl, while the other, not my own case, was a lunatic. In both the operation was followed by meningitis and death.

CONCLUSIONS

We are all aware of the famous reproach to otology that ear diseases are divided into two classes: first, those that anyone can cure with a syringe; and, second, those that nobody can cure and that should be sent to an ear specialist. This witticism is, of course, unfair, but there is just enough truth in it to give it a sting. We know that we can deal more or less successfully with suppuration in the middle ear and its consequence. Why cannot more be done for non-suppurative deafness?

The following questions suggest themselves for solution:

(1) Is otosclerosis a pathological entity?

(2) Is it congenital? Is it ever present in the labyrinth capsule of the foetus or infant? A co-operative investigation of the inner ear in a large series of cases of foetal death from placenta previa or breach presentation would settle this point. Manasse has examined 200 foetuses and in eleven found symmetrical islands of cartilage in the labyrinth capsule in the site of election. Is this a preliminary to otosclerosis? I have heard Gray say that in his opinion one person in every 200 suffers from otosclerosis, but if Manasse's observations are accepted, we should expect a percentage of 5.5.

(3) Is otosclerosis an inflammatory affection and does it follow otitis media? Can it be distinguished sharply from middle ear catarrh?

(4) Is otosclerosis a degenerative or wasting disease?

(5) Is there any connection between disorders of the endocrine glands and otosclerosis?

(6) What is the cause of deafness in otosclerosis seeing that in early cases at any rate there is no bony ankylosis of the stapes?

(7) What is the connection, if any, between otosclerosis, nerve deafness and congenital deafmutism?

(8) How often is the vestibular apparatus involved in otosclerosis and why is there giddiness in one case and not in another?

If an investigation is organized I suggest that it should be divided into two parts:

(A) *Laboratory work*—microscopical, chemical, experimental.

(B) *Clinical.* (1) Statistical: as regards age, sex, heredity, distribution, association with other diseases.

(2) Symptoms, clinical examination, diagnosis.

(3) Treatment: medicinal, ductless gland therapy, vaccines, local non-operative procedure, operations.

The two parts of the investigation should, of course, be co-ordinated, e. g., the clinical examination and post-mortem microscopical examination.

Individuals have different gifts and have been specially trained along various lines. Some are clinicians, others laboratory workers; some of the latter have been trained as chemists, others as physicsts or pathologists; some lean toward medicine, others to surgery; one man laboriously attacks a problem from the pathological and microscopic point of view, another has a "brain wave" and is carried to a brilliant, and let us hope, a correct conclusion. My suggestion is that in each country the problem should be tackled by a team composed of men who individually and together would represent all these different aspects.

A co-operative investigation can only be originated in America, which is the most universally popular—perhaps I should say the "least unpopular"—country in the world.

THE STENGER TEST FOR THE DETECTION OF SIMULATED UNILATERAL TOTAL DEAFNESS.

Dr. Matthew F. Czubak, Wheeling W. Va.

In all the modern text books on Otology, considerable space is devoted to the detection of malignerers simulating unilateral deafness and emphasis is laid upon the difficulties encountered. The test which seems to be the easiest of performance and most infallible in its results has received no mention at all. While a student at the Graduate School of Medicine of the University of Pennsylvania, the writer was taught the Stenger test by Dr. George W. MacKenzie during his sojourn as a lecturer there and it is to him that the credit is due for the information given in this article.

The test is made with a pair of small A Bezold-Edelman tuning forks of 654 D. V., unweighted and synchronized to vibrate in sympathy with each other. Thus, if one fork be struck a most delicate blow and then be held near to the second fork, the latter will also vibrate without having been struck. The carrying distance of the forks used is so short that the tones from a vibrating fork applied to one ear can not be heard by the opposite ear no matter whether the former ear be normal or deaf. The principle underlying the test is that if an individual is tested with two forks of the same pitch held at equal distances from the two ears, the sound can not be lateralized. If one fork is held at a shorter distance from the first ear than is the other fork from the second ear, only the more intense sound will be heard. The intensity of the sound is in inverse ratio to the square of the distance. Thus, if the first fork is held one inch from the right ear and the second fork is held two inches from the left ear, the sound at the right ear will be four times as intense as that at the left ear. The sounds referred to are the normal vibrations of the forks and not the overtones which are produced by striking the forks against some hard object. The overtones do not adhere to the above mentioned laws of physics.

FIRST STEP.

To facilitate the explanation, let us assume that we are examining a patient who is simulating absolute deafness in his right ear, though actually his hearing is normal and equal in the two ears. The patient is seated in a chair with his back to the audience to be convinced of his malingering. The examiner stands behind the patient and holding one fork in each hand, strikes the two forks an equal blow against his thigh or some other cushioned object which will

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produce no overtones. The forks are then placed at equal distances from the two ears (about two inches) and the patient is asked in which ear he hears the louder sound. Being on his guard, he will invariably answer, "In my left ear". The admission that he hears one of the vibrating forks with his left or good ear is essential, especially if there be an audience present.

SECOND STEP.

The forks are again placed in vibration. This time, the right fork is placed about an inch from the right ear while the second fork is held about two inches from the left ear and the patient is asked, "And now in which ear do you hear the louder sound?" As explained above, the sound of the right fork is four times more intense than that of the left fork and the two forks being of equal pitch, only the louder of the two sounds is actually being heard. He may give himself away this early or may continue to lie systematically. If he answers that he hears the sound in his left ear, the left fork is moved farther and farther away from the left ear and at each new distance the question is repeated, "And now?" If he does not become confused by the increasing difference in intensity of the two forks, the third step is in order.

THIRD STEP.

The forks are struck again, preferably in view of the patient in order that he may see that the two forks were struck and not just the one. At the same time the examiner admonishes the patient to answer promptly as soon as he hears the words, "And now?" Stepping quickly behind the patient, the examiner silences the left fork by smothering it in his hand. The two forks are then placed at equal distances from the two ears and the question is asked, "And now?" To the patient's mind, conditions are exactly as in the preceding step, for he hears only the right fork as before and has no knowledge of the fact that the left fork is not vibrating. If this time he answers that he hears the sound in his left ear, he is caught, for the left fork is not vibrating and he can not hear the right fork with his left ear no matter what the condition of hearing is in either ear. If he answers that he hears it in his right ear, he will have told the truth. If he insists that he hears no sound at all, the last step is in order.

FOURTH STEP.

Standing behind the patient, the examiner says, "Let's try that one over again" and strikes the forks equally. The left fork is now held at about two inches from the left or good ear while the right fork is simultaneously brought to a position about one inch from

the right ear. The question is again asked, "And now?" He actually hears only the right fork for the third time in sucession and believes the last three steps to be the same procedure repeated. Having answered the last time that he did not hear the sound at all, he must be consistent and answer the same this time. The examiner then moves the right fork entirely away from the right ear but continues to hold the left fork in its original position and asks the usual question. The patient now notices that the sound has shifted from the poor ear to the good ear and is in a quandary. No matter what his answer, he is trapped. If he answers that he hears it with his left ear, which is the truth, he must have lied the previous time he responded, for if the right ear were really deaf, only the left ear would have heard any sound and he would have so answered instead of saying that he heard no sound at all. If he answers that he hears no sound, he likewise must be lying, for did he not originally admit to hearing in the left ear when the fork was held at the same distance? (First Step). If he answers that he hears with the right ear, he will have confessed his malingering and at the same time will be telling a lie for this time he could not have heard it with his right ear.

SUMMARY.

If, during any part of the test the patient admits hearing with his right ear, no more need be done. If he claims that he hears the sound with his left ear, the second and third steps eliminate the improbability of that reply. If he insists that he hears no sound, the first and last steps disprove that statement. If the patient does not claim absolute deafness, the amount of hearing lost can be estimated roughly by measuring the distances at which the two forks must be held to make the sound heard in one ear and then in the other, bearing in mind the law of the intensity of sound being in inverse ratio to the square of the distance. The percentage loss of hearing can be more precisely figured, however, by the use of the standardized tuning fork and the stop watch.

CONCLUSIONS.

1. The Stenger test is particularly adapted to the detection of malingerers simulating unilateral absolute deafness.
2. It is easily and quickly performed and requires no expensive apparatus.
3. It can be done before and explained to any audience in any place.
4. It seems to be infallible.

75 12th Street.

SUSPECTED MASTOIDITIS: CLINICAL DIAGNOSIS WITH SPECIAL REFERENCES TO THE INTERPRETATION OF THE X-RAY PICTURES.*

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In the majority of cases of acute mastoiditis, the clinical signs are evident. In a small minority, either no clinical signs are present or else they are so variable or so complicated by other symptoms, that it is almost impossible to make a diagnosis. In this paper, it will not be my object to go into the usual signs and symptoms which are present in the majority of cases because it will be taken for granted that these are known to every otologist. However, within the course of a year, any otologist who has a large clinical experience, will be brought in contact with a number of cases in which there is a suspicion of mastoiditis and in which it is impossible to make a definite diagnosis. Even with all the usual facts at hand, it is impossible for one to definitely state that the mastoid is so involved as to warrant operation.

The usual signs and symptoms of acute mastoiditis will vary considerably and it is often a question whether one may be guided definitely by them so that he can state that the patient should be operated upon or not. I am inclined to feel that a great many cases of suppuration of the middle ear with mastoid symptoms are unnecessarily operated upon. Conversely I feel that there are a great many cases which should have been operated upon at an earlier date, where operation has been delayed until some complicating factor has occurred which has made the operation far more serious than it originally would have been.

The presence of acute tenderness over the mastoid with radiating pain and tenderness and a suppuration from the ear canal, is not sufficient to establish a diagnosis of mastoiditis. Even where there is sagging of the posterior wall with the above symptoms, one cannot definitely say that it is impossible for the patient to get well without operation. I have in mind a report which I made to this society over ten years ago. I had six patients in one family who developed acute suppurative otitis media at one time, four of them developing the clinical signs and manifestations of acute mastoiditis. The temperatures of these patients ran as high as 103, and

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104. One of them was operated upon but the other three absolutely refused; yet all four got well. I have no doubt that many cases that show such acute symptoms as these and refuse operation, get well in the course of time, although naturally one is running a great risk. One has to consider that in such cases, the operation is the conservative measure rather than the radical one.

Let us cite two hypothetical cases. In one instance, the patient suddenly develops a severe pain in the ear. An incision is made in the drum within a short time. There is profuse discharge of serum or pus. The pain and tenderness over the mastoid process gradually increases within the course of twenty-four to forty-eight hours. There is no doubt that there is an inflammatory condition present in the mastoid cells. The temperature rises to 102-103 and the patient is very much distressed. There is a throbbing feeling within the ear. At the end of that time, the symptoms remain stationary and the patient remains in this state for three to four days and finally the symptoms retrogress until, in the course of a week, the tenderness has practically disappeared and all that remains is a slight amount of discharge from the ear which gradually clears up. This patient, no doubt, had an acute inflammatory condition of the mastoid cells which did not go on to an actual suppuration and the drainage was sufficient through the incision in the drum membrane. Now there is no doubt that such a case was one of acute inflammatory mastoiditis but is of the type which readily takes care of itself without operative interference. In other words, acute inflammatory conditions of this kind will very frequently get well if Nature is allowed to do her part. Let us take the opposite type of case. A patient has an incision made in the ear drum or there has been spontaneous perforation. A profuse discharge of thick pus takes place. The temperature is low or there is none. There is very little tenderness over the mastoid and practically no pain. The discharge continues for a considerable length of time and finally the canal becomes more narrowed and there is a sagging of the postero-superior wall. At no time does the temperature rise above 100 and at no time is there any real acute tenderness over the mastoid bone. There might be tenderness at the tip on deep pressure. In the course of time, the discharge may or may not become lessened and in many instances remains of the same type for three to four weeks. The hearing is considerably diminished in this ear. In other words, the patient has gone into the sub-acute or latent stage and the probabilities are that the disease will remain stationary for a considerable length of time, until some complicating condition arises. Such a

case is far more dangerous than the former one. Eventually, such a patient must be operated upon. Between these two types, one sees all sorts of cases which vary from the moderately acute case to the type which presents no symptoms.

From the foregoing, one may say that the cases of acute mastoiditis divide themselves into two classes: one, the apparent case, which can be easily diagnosed, and the other, the suspected case. We shall have nothing to do with the apparent cases in this paper and only dwell on the suspected cases where the symptoms are so variable that it is impossible to make a definite diagnosis in many of them without taking all signs and symptoms into consideration.

At this point, it might be wise to ask what one considers acute mastoiditis. Is it the actual pathology within the mastoid cells one should think of or should he look at the case mainly from the operative point of view? To my mind, there is seldom an acute suppuration from the middle ear, which lasts over two or three days, in which there is not some involvement of the mastoid cells. There may be only an inflammatory reaction or there may be an acute retention of pus within some of the cells, many of which will drain through the incision in the drum. In other words, in almost all instances in which there has been a discharge from the ear, over a period of three to four days, one may definitely say that there is a mastoiditis present; yet it would be unwise to make such a statement to the patient or to the patient's family, for the only consideration that one should have is whether the mastoid involvement is such to warrant operation. In other words, in the majority of cases, one must consider mastoiditis mainly from the operative view point. I have repeatedly stated to patients that the mastoid involvement is present in almost every case where there is discharge from the ear which has lasted more than four to five days and in which there are evidences of mastoid involvement such as temperature, malaise, tenderness over the mastoid bone, etc., etc.; and yet, I never consider mastoiditis from any other point of view than the operative one. One frequently hears of patients who say that they have had mastoiditis which recovered without operation and therefore they have advised patients, who are in a similar condition, not to have an operation performed. Such advice is just as much warranted as the statement that because one patient had appendicitis and was not operated upon, another should not be operated upon. In other words, the surgeon, although he may make a diagnosis of appendicitis and state that the patient has gotten well without operation, in the majority of instances he seldom makes such a statement unless he considers

it from the operative point of view. This does not take into consideration those cases of appendicitis of the sub-acute or chronic variety which cause intestinal stasis.

How shall we interpret the diagnostic symptoms and signs in cases of suspected mastoiditis? In order to dwell upon this in a definite sort of a way, the symptoms should be divided into the following and each one will be taken up under its separate heading:

1. General symptoms, such as headache and malaise.
2. Temperature, pulse and respiration.
3. Pain and tenderness with edema over the mastoid and the involvement of the glands below the mastoid process.
4. Discharge from the ear canal, amount and the character, organism which is present.
5. The narrowing of the canal.
6. The prolapse of the drum or the sagging of the postero-superior wall.
7. The pulsation of the opening of the drum.
8. The evidence obtained on suction on the drum.
9. Blood picture.
10. The value of the X-ray picture.

1. *General symptoms, such as headache and malaise.* As a rule such symptoms are present in the majority of cases until a sub-acute stage is reached when they may entirely disappear. The general physical condition of the patient is usually far below normal. I consider that a radiating pain of the hemi-carnial type, on the same side as the ear disease should warrant some consideration. In other words, wherever there is a radiating pain along the supra-orbital nerve or over the temporal region, there is evidence that there is some involvement of the mastoid cells overlying the superior part of the middle ear and running forward into the zygoma. Generalized headache is not of as much significance, although I have seen patients who have complained of very severe headaches, which could not be localized and which suggested some intracranial condition.

2. *Temperature, pulse and respiration.* To my mind, the temperature is not of a great deal of importance, unless it persists after the third or fourth day. One may naturally expect a high temperature at the onset of the trouble, particularly before a large opening is made in the ear drum, but it is surprising to see how many times the temperature is very low, seldom going above 100 or 100½, and in many instances, the temperature is absolutely normal. I recall one case, a child who had a discharge from the middle ear for three

to four weeks. During the first three weeks, the temperature ran between 100 and 100½ and then it became normal. You can imagine of the chagrin of the parents, when, in the fourth week, I stated that the child had to be operated upon. Within a few days, a definite change had taken place within the canal and in the middle ear. There was a narrowing of the canal, with a prolapse of the posterior wall. The diagnosis was definitely corrobrated by the X-ray picture and by the operation which took place within the next twenty-four hours. Very often patients will be seen who have run no temperature for a considerable length of time and yet have such a virulent type of mastoiditis that there has been an entire destruction of the mastoid with an exposure of the sinus and, sometimes, of the dura. Of equal significance however, is the lack of temperature for a considerable time and then a sudden rise of temperature to 101 or 102 of a persistent type. Such a temperature usually means a reinfection of the mastoid cells. In other words, a certain part of the mastoid has originally been involved, but there has been proper drainage. Then suddenly, other cells, probably those posterior to the sinus plate, become involved, with the result that there is a rise in temperature due to sudden retention within these cells.

3. *Pain and tenderness.* The pain within the middle ear itself and over the mastoid is often significant, especially after the first few days. Often there is a throbbing sensation or pulsation which persists and sometimes is syncronous with the heart beat. I consider pain only of significance when it is associated with definite tenderness; for the pain will vary considerably according to the type of individual one is treating. However, when pain is associated with definite tenderness over the mastoid, one has to take it seriously into account. The tenderness is often persistent over the antrum and extends further back. In other words, tenderness is only of definite worth if one notes whether it is gradually progressing until he is able to state that, in his own mind, he feels that the cells behind the sinus are involved. Yet, how many cases we see where there is absolutely no tenderness at all and sometimes no pain! At this time, I can call to mind any number of patients who never had any tenderness over the mastoid proper and when operation was performed, the most virulent type of mastoiditis was found. The lack of tenderness may be due to any number of factors, chief among which are the thickness of the cortex of the mastoid bone, the amount of drainage which takes place from the middle ear and the lack of susceptibility of the patient to pain. As I said before, when tenderness is present, it has a certain significance, if it can be

observed from day to day but lack of tenderness may mean nothing. Of a great deal of importance in these cases, however, is edema over the mastoid process or a feeling of thickening of the tissues. The amount of edema is often so slight that it is questionable whether any is present or not and the comparison of the tissues, by placing the finger tips over the mastoid bones on both sides, must be made, in order to see whether there is a different sensation imparted to the fingers. Often edema can be determined, particularly in young children, by noticing the difference in the fold between the ear lobe and the mastoid bone on the two sides; yet one must take into consideration the fact that in many of these cases, there are small glands present over the mastoid process which may be taken for edema or which may give rise to a good deal of tenderness. There have been instances, within the past two years, in which the only significant sign was the presence of this peculiar feeling to the finger tips which allowed one to determine the difference in the amount of tissue between the skin and the mastoid bone on the two sides. Glands over the mastoid process or below it may complicate the picture. One must always consider whether these glands are the cause of the temperature and the tenderness over the mastoid or whether they interfere with the lymphatic circulation of the mastoid proper.

4. *Discharge from the ear canal.* The amount and character of the discharge have definite significance in all suspected cases of mastoiditis. In a certain few cases, the discharge remains serous during the entire course of the disease. The interpretation that can be put upon the discharge will depend upon numerous factors, the most important of which to my mind, is the bacterium which is causing the trouble. I cannot impress too strongly the necessity of making bacterial cultures of all discharges, particularly when one makes the primary incision in the drum; for at such time, one may be able to get a pure culture where one cannot get it at a future date. The presence of a staphylococcus or a pneumococcus ordinarily means that the disease can run on for a considerable length of time without causing serious trouble. This eliminates, however, the pneumococcus type three, which was formerly called the streptococcus mucosus capsulatus. If a streptococcus, particularly the streptococcus viridans or the streptococcus hemolyticus is present, in all likelihood there will be a progression of symptoms which will lead to operative interference. The admixture of mucus in the discharge may incline one to the feeling that the discharge comes mainly from the naso-pharynx. But, if other evident signs of mastoiditis are

present, one must not place too much reliance on this one phase of the middle ear inflammation.

5. *Narrowing of the canal.* Of all the significant symptoms, I believe that narrowing of the canal is of the most importance. This is particularly so if there is no inflammatory condition of the canal or any furunculosis. The canals of both ears should be compared at each examination and any evidence of narrowing should meet with a suspicion that there is some pressure behind the posterior wall which is causing it to bulge forward. In one of the instances which I shall cite later, there was a considerable narrowing of the canal, without any other symptoms, and the diagnosis of mastoiditis was only made after an X-ray picture was taken. In another case, there was absolutely no evidence of mastoiditis other than an absolute stenosis of the canal due to pressure on the posterior wall.

6. *Prolapse of the drum or sagging of the postero-superior wall.* This is considered a diagnostic sign in the majority of cases. However, in numerous instances, I have seen mastoid disease clear up where there has been considerable sagging of this posterior portion of the drum. Pressure of this kind can be caused by a filled up antrum, which is draining nicely through the incision in the drum and if other symptoms do not warrant an exploration of the mastoid cavity, this, in itself, should not be taken into account. However, in suspected cases, where there are any associated symptoms, such as fever, or narrowing of the canal or tenderness over the mastoid, one must consider that the symptoms or sign of most diagnostic importance, is this prolapse of this posterior portion of the drum.

7. *Pulsation of the opening in the ear drum.* Of equal significance, in the majority of cases, is the pulsation of the opening in the drum. If this persists for a considerable length of time, it is indicative of a retention behind the drum. This does not always mean that a further incision is necessary. I have seen the same amount of pulsation when repeated incisions in the drum have been made. Such a pulsation is easily seen through an ordinary speculum or through an electric otoscope. However, it should only be considered of significance if it is associated with other symptoms which show a progression of the trouble.

8. *Suction on the drum for retention.* In the majority of cases, the perforation in the ear drum, narrows itself down to a minute opening and it is often surprising that a proper amount of discharge can take place through this opening. It is an excellent procedure to use a certain amount of suction through this opening daily, to see how much retained secretion can be brought out by this pro-

cedure. Sometimes, there is very little discharge from the perforation and one thinks that the condition is clearing up and yet if he will attach an electrically lighted otoscope, with magnifying glass and rubber tubing, through which suction can be exerted, often he will see that there is a great deal of retention behind the drum. When there is such retention especially if associated with other symptoms, it means that there is not the proper elimination of the diseased factors in the middle ear and perhaps in the mastoid itself.

9. *Blood examination.* The blood examination, in the majority of cases of acute suspected mastoiditis, may show little or nothing. In the majority of instances, when there is free discharge from the ear canal, the count is normal, but where there is any retention, particularly in the mastoid cells, there is usually a rise in the total white blood count, to twelve or fourteen thousand and the relative number of polynuclear cells is increased. The increase may not be more than to 78 per cent to 80 per cent. If it rises above that, it should be considered gravely although one should expect a higher blood count in children than in adults. One blood count, by itself, may mean nothing but repeated blood counts are of value if they can be compared, and it is always wise to correlate the blood picture, with the other symptoms.

10. *Value of X-ray pictures.* Since we have been able to examine the mastoid process carefully, by properly taken X-ray pictures, we are in a much better position to decide exactly what pathological condition is present. It is of the utmost importance that a well trained roentgenologist takes these pictures as such pictures are open to much misinterpretation if they are not taken in the right way. The X-ray picture will often show a cloudiness on the diseased side as one well knows, and one should examine carefully to see if there is any breaking down of the septa between the various cells. At times, it is necessary to take more than one X-ray picture and sometimes it is necessary to check up on pictures taken by one man by having them taken by another man. In the majority of instances, when clinical symptoms are present, which point towards an acute process, an X-ray picture showing cloudiness of any kind is significant of a diseased process which warrants operative interference.

However, I hesitate to state that the X-ray picture should make the diagnosis. By the citation of three cases, I shall show you that in suspected cases, or even in unsuspected cases, where there has been a previous discharge from the ear, the X-ray picture is of the greatest value. On the contrary, I have had many

pictures taken where there has been an acute process lasting only a few days, and in other cases where the process has gone on for a number of months, in which a definite cloudiness was present in the X-ray picture and yet for one reason or another, no operation was performed and the patient eventually recovered. What will happen to these patients in the far future, it is impossible to say. If one feels that the ear condition is clearing up and that the hearing is returning to normal, he is inclined to be cautious and not operate, simply because an X-ray picture has shown a certain amount of cloudiness. One instance of this kind will suffice. A short time ago, a man was referred to me who had had a discharge from the left ear for three weeks. A culture from the discharge, which was profuse at the first examination, showed a long-chain streptococcus. There was a great deal of pulsation to the drum opening and there was considerable sagging of the postero-superior wall. There was absolutely no tenderness over the mastoid either on tapping or on pressure. I was inclined to feel that the man had an acute mastoiditis but as he had no temperature, I was perfectly willing to observe him carefully. After observing him for a week, and seeing that there was no difference in his condition, I insisted that an X-ray picture be taken. The report from the laboratory showed cloudiness of the entire mastoid bone on the diseased side. There was no breaking down of the septa between the cells. I told him, that if he were not better in the course of two or three days, it would be necessary to open up the mastoid bone. He came back to me at the end of the third day, with absolutely no discharge from the middle ear, with a perforation that was healing up, and with an ear drum which, although slightly thickened, showed a return to normal. There still was no tenderness or pain over the mastoid and no temperature. At the end of another week, the hearing had returned to normal and the patient was apparently cured. This is only one of many cases. I feel if an X-ray picture were taken of all mastoid processes during the first few days, when the acute symptoms were present, particularly at the time when the patient was suffering pain in the ear, in all likelihood, we would find a cloudiness of the mastoid in almost every case. Moreover I feel sure if we again take a picture at the end of a week, when the symptoms are retrogressing we would find that the entire process had cleared up and that there was no evidence of any disease which would necessitate our exposing the patient to an operation. In other words, we are in the same position in regard to the X-ray pictures of the mastoids as we are in regard to X-ray pictures of the other cavities of the

head. The X-ray picture is only of value when it is taken in conjunction with the clinical symptoms. Yet, bearing all these facts in mind, every once in a while, a case comes to light, in which there is absolutely no manifestation of any diseased condition in the middle ear or mastoid, and in which X-ray pictures show a diseased condition which warrants operation. I shall briefly cite three cases which are of exceeding interest:

Case 1. A patient, 34 years of age, who came to me two years ago having complained six weeks before that he had a slight discharge from the ear which lasted twenty-four hours. When he presented himself to me, he had a narrowing of the ear canal which was not sensitive to pressure and the drum could not be seen. However, the ear could be inflated without any trouble. The hearing could be considerably improved, particularly when a small speculum was inserted in the canal. There was no tenderness of any kind over the mastoid and no temperature. The patient complained of a fullness, which was indescribable, on that side of the head. After making numerous incisions in the canal, to see whether the stenosis could not be overcome, I advised him to go away for two weeks, at the end of which time he returned, still complaining of the same symptoms and then I stated that, in order to relieve him from worry, it might be advisable to have an X-ray picture taken, which I felt sure would be negative. I was considerably surprised to have Dr. Dixon state to me that the entire mastoid process was destroyed. After consultation with Dr. Robert Lewis, we decided to do an exploratory operation and this was performed the following afternoon. I was greatly surprised, when I opened the cortex of the mastoid, to come upon a large abscess cavity, with a peri-sinus abscess, with every cell completely destroyed. The cells were thoroughly exenterated and the wound closed except at the lower angle. The patient was entirely well at the end of ten days.

Case 2. A little child was seen by me about two months ago. Three weeks previous she had had a mild influenza and then developed scarlet fever. At this time was a slight congestion of the ear drum without any discharge. She was under the care of a competent pediatrist, who claimed that the child was in excellent condition with no temperature, and no complaint whatsoever. However, at the end of three weeks, when he called one afternoon, he found that the child had a temperature of 103 with a gland about the size of a small egg, below the tip of the mastoid. I was called in consultation and found that, although the right ear canal was patent, the left ear canal was so stenosed that it was impossible to see the drum.

There was no tenderness whatsoever. Palpation over the mastoid elicited a certain amount of tenderness near the tip which coalesced with the gland below. However, on the strength of my previous experience with the above case, I insisted that the patient had an acute mastoiditis; the diagnosis of which was corroborated by X-ray picture that evening and by consultation with Dr. Fred Whiting. The following afternoon, the child was operated upon. The entire mastoid cavity was filled with pus, with a peri-sinus abscess present and an exposure of the dura. The mastoid cells were thoroughly exenterated. At the end of about two weeks, the patient had entirely recovered. In this case again, there was absolutely no evidence of any ear condition and the diagnosis could not have been definitely made without the X-ray picture.

Case 3. A young woman, came to me about two months ago, complaining that about five weeks previous she had had a slight discharge from the ear canal for two days. At the time of the examination the ear canal was found normal, the middle ear was clear and hearing normal. There was no tenderness over the mastoid but the patient had a peculiar irritating pain over this region. After eliminating all other causes for the condition an X-ray picture of the mastoid was taken which showed general cloudiness. She then consulted Dr. Fred Whiting who advised that she be observed, but a few days later, slight tenderness developed over the tip of the mastoid and another consultation was held with Dr. Robert Lewis and we put it up to the patient to have an exploratory mastoidectomy. She was operated upon the following day. An extremely thick cortex was found. It was necessary to chisel deeply down into the mastoid bone before any cells were found. There was no evidence of any pus, but on extending the search down towards the tip of the mastoid one entered a very large cell, filled with necrotic bone and caseous material, and exploring upwards, one found that this connected along the posterior wall with the antrum. This cavity was thoroughly cleaned out. Nothing else of a pathological nature being found, a small gauze drain was put in the antrum and the wound closed. The wound healed within four days.

How can we account for such cases? Only in one way. No doubt at the time of the acute process, an inflammatory condition occurred in the mastoid cells as well as in the middle ear. The attic particularly, being small is closed off from the middle ear cavity, with the result that we have an empyema of the mastoid cells, which shows no evidence until sufficient destruction takes place. In all these three cases, the diagnosis could not have been made and ap-

parently could not have been suspected, until an X-ray picture was taken. I hesitate very much to state that I would operate upon the findings of an X-ray picture but if one has a definite correlated set of symptoms which lead him to feel that the patient has some trouble which he cannot overcome, he must take into account the fact that a possible discharge from the ear a short time before may allow of some retention of infected material within the mastoid, which will give no evidence. Certainly in none of these cases was there any evidence in the middle ear or on the drum to warrant our considering the question of an acute process within the mastoid, yet in two of these cases, there was a considerable narrowing of the ear canal and in the third there was a dull feeling behind the ear with finally an acute tenderness over the mastoid.

In conclusion, one may ask the question, "What would you consider the most significant signs and symptoms of suspected mastoiditis?" In the order of their importance, I should suggest the following:

1. The character of the discharge and type of infecting organism if discharge is present.
2. The narrowing of the canal wall, which cannot be accounted for by any acute symptoms within the canal itself.
3. The pulsation of the opening in the drum showing retention.
4. The prolapse of the drum with the sagging of the postero-superior wall.
5. The amount of retained secretions that can be eliminated from the middle ear by suction.
6. The general physical condition of the patient, including symptoms such as headaches, malaise, temperature, blood picture, etc.
7. The interpretation of the X-ray picture.

22 W. 74th St.

FIVE ATYPICAL CASES OF MASTOIDITIS IN CHILDREN.

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The five cases of which we report were cases that entered the ear, nose and throat department of Grady Hospital between March 10th and April 11th, 1923, a period of one month and a day. All of the cases presented the same symptoms and appearance, all were operated on, three dying and two living and the two living being in good health at the present time.

All cases entered in a semi-comatose, lethargic condition, and were as near skeletons as one could imagine a human being. All gave a history of influenza of five to fifteen days previous, with a progressive weakness and emaciation. The cases entered the hospital March 10th, 23rd, April 1st, 10th and 11th respectively. The first two were brothers. The onset of all cases were given as severe headache and pain in upper abdomen with a severe cold and "achy feelings" all over the body. All cases were brought to the hospital at the development of the lethargic state. All cases were negroes.

The ages of the children were five, six, seven, ten and fourteen years respectively. None gave any history of ear ache or running ears. As to history nothing else of importance is worth while mentioning.

As to the physical condition, all entered in a lethargic state, with extreme emaciation and would answer questions if sufficiently aroused. None showed any signs of any cranial nerve paralysis or weakness and their neurological symptoms were negative except the lethargic state they represented. The eye grounds were negative. The only other positive point in their physical examination was that the ear drums were bulging in each case, but with no tenderness at any place over the mastoid region or middle ear. The drums of all were widely incised with resulting pus, examination of which microscopically showed streptococcus (non hemolyticus) staphylococcus and pneumococcus. At no time during their stay in the hospital was there any mastoid tenderness or pain. Their pulse ranged from 132 to 150 and their temperature in no case was affected by the myringotomy. X-ray of all mastoids in all cases showed them cloudy and diseased. The white cell count varied from 10,000 to 20,950 with polymorphonuclear leucocytes ranging from 47 to 78 per cent. Small mono-nuclears from 15 to 37 per cent. Large mono-nuclears from 2 to 6 per cent, and transitionals from 2 to 7 per cent.

The spinal fluid, the urine, blood and spinal Wassermann,

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stools, blood culture, widal, sputum (T. B. Vicints), and X-ray of lungs were all negative in each case.

As you will see the only positive points we had were the lethargy emaciation with fever, fast pulse, and bulging ear drums with resulting pus after myringotomy (but no drop in temperature following same) with a leucocytosis and an X-ray report showing mastoids cloudy and diseased.

The first case that entered the hospital was thoroughly studied for fourteen days and every diagnosis from Alpha to Omega suggested, but with no one final diagnosis. Finally it was decided by the pediatricians and otologists to open the mastoids on the X-ray diagnosis and evidence of running ears over a period of two weeks. A double mastoidectomy was done under local anesthesia and an extremely advanced and widely spread purulent mastoditis found on both sides.

All the other cases were the same and were likewise operated on under local anesthesia, the findings in each case showing an advanced purulent mastoiditis.

Of the five cases three died and two are living, well, and fully recovered. Of the three children that died an autopsy was obtained on two of them, the findings of which grossly and microscopically showed no evidence of pathology anywhere except in the mastoid and middle ear.

The treatment used in all cases was that of stimulation and forced fluids. The two cases that are living are the ones that were operated on early after their arival into the hospital.

Conclusions:

1. That mastoiditis following influenza may be severe, without giving all of the typical symptoms.
2. That the symptoms may appear as one of a general constitutional disease.
3. That extreme emaciation and lethargy may accompany an undiagnosed mastoiditis, progressing even to death.
4. That the pus coming from the middle ear of all the cases cited did not pulsate and was under no pressure.
5. That no history of ear ache or running ears was given. That no mastoid tenderness was elicited at any time.
6. That both mastoids were involved in each of the five cases.
7. That X-ray is of value in these obscure cases.
8. That none of these cases were of a streptococcus haemolpticus variety.
9. That early operation is the proper procedure.
10. That a complete mastoidectomy can be done under local anesthesia with very little pain or shock to the patient.

A MASTOID BANDAGE.

GEORGE M. COATES AND BENJAMIN H SHUSTER, Philadelphia.

No originality is claimed for the mastoid bandage herein described. It is, in fact, rather a combination of ideas worked together and developed during many years experience, and in its present form has proved most satisfactory and reliable. As we have nowhere seen any printed description of a bandage employing these principles and as we have taught this method of holding the mastoid dressing in place to many students, internes and nurses,

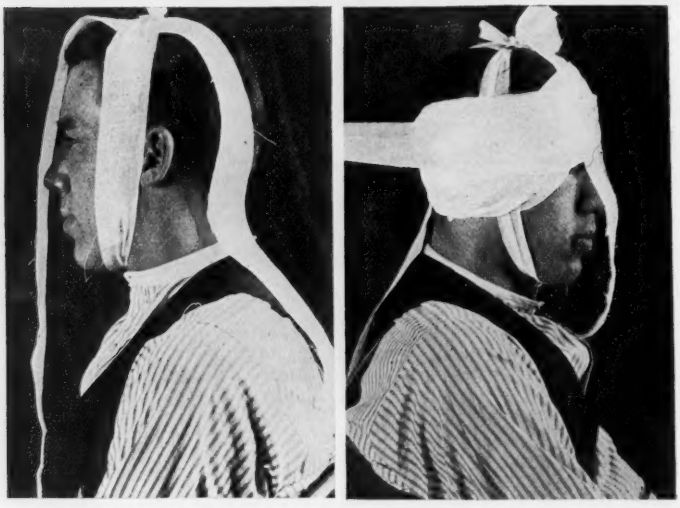

Fig. 1.

it seemed possible that there might be enough of interest to otologists who were not familiar with this method of bandaging to warrant its brief presentation.

Everyone is familiar with the difficulties a beginner has with the mastoid bandage as usually employed and as described in the text books. It is seldom neat, seldom securely holds the mastoid dressing firmly in place, gives way to prying fingers, and has to be reinforced by numerous strips of adhesive plaster or safety pins. A fractious adult or an irritable child makes its smooth application a

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matter of considerable skill unless the eye is included and the lower end of the dressing left dangling and uncovered. The child easily pushes it upward to scratch underneath or pulls it downward over the forehead and eyes. If pins are used to fasten it, they sometimes prick the skin or work loose; if adhesive is used it prevents unwinding the bandage, which must be cut away for redressing, and it not infrequently sticks to the hair. In the bandage as applied on our cases, neither pins or adhesive plaster are employed. It can be applied as well to a double mastoid as to a single one.

With the mastoid dressing in place, a strip of gauze bandage, about a yard long, is placed over the head in the vertical line, be-

Fig. 2.

ginning below the tip of the nose, with the tail hanging midway between the shoulders. Another piece of gauze about two feet long is placed under the chin and tied loosely with a single knot on the middle of the top of the head (Fig. I). The remainder of the bandage is now used to hold the dressing in place, being applied in the usual way, but without reversing, care being taken to place it smoothly and to cover both the upper and lower limits of the gauze dressing. It must not be applied tightly, however, and not very many turns are needed. No care need be exercised to keep it away from the eyes as is usual in this form of bandaging and wherein the greatest difficulty is often encountered (Fig. II). When this

part of the bandage has been applied to the satisfaction of the dresser, the second piece of bandage is cut beneath the chin and untied at the top. We now have two short strips of bandage, passing under the circular turns of bandage at about the outer canthi of the eyes. Each of these strips is, in turn, tied over the forehead turn of circular bandage, thereby lifting the latter away from the eyes and eyebrows, tightening them and making the whole bandage firm and secure (Fig. III). The loose end of the circular bandage is now tied to one of these loose tails. A bandage properly applied in this manner can be dislodged upward only with considerable difficulty. But it can still be pulled downward over the eyes. To obviate this, the two ends of the vertical strip are now tied on top of the head. This prevents the bandage slipping or being dislodged downward. The knot must be well toward the top of the head so that it will not cause discomfort by pressing on the skull when recumbent, no matter in what position the patient lies. All the loose ends of bandage are now cut away with scissors (Fig. IV).

For removal, the bandage may be cut through over the forehead in the usual way, or the retaining strips (figures I and II) may be cut and the entire bandage unwound to be used again if desirable.

Such a bandage may be made as neatly by a beginner as the usual bandage by an expert, if a little care and patience is exercised. We feel that it should be a matter of pride to every dresser to place all bandages neatly, smoothly and exactly, and when so done, a good deal of comfort accrues to the patient. A sloppy dressing should never be tolerated and adds greatly to the risk of secondary wound infection. This bandage can be readily modified to apply to almost any form of head dressing.

1811 Spruce St.

STUDY OF SINUSITIS IN CHILDREN, PRELIMINARY REPORT.*

DR. JOHN M. LORE, New York.

At the Manhattan Eye, Ear and Throat Hospital, under the supervision of Dr. Samuel McCullagh, a survey of the problems in the study of sinusitis in children has been attempted. Early in this study we realized that the problem was a good deal more complex than one was inclined to believe.

According to Dr. Dean of Iowa City, who probably has done more systematic work on this subject, from 70 per cent to 80 per cent of the cases of sinusitis in children get well after a tonsil and adenoid operation is performed. It is the problem presented by the 20 per cent or 30 per cent of cases which do not get well after this operation—that we are interested in.

These cases are ambulatory and represent the same type which comes to our office. It is difficult to hospitalize these cases; first because the parents are loath to leave their children in hospitals, and secondly because hospital facilities are taken up with more urgent cases.

A careful history of these cases is of importance; particularly as to the relationship of the exanthemata and influenza to the onset of this condition. The question of diet, habits and environment is also of importance.

Another question which arises is, "What relationship exists between anatomical defects of the nose and this disease?" Early in this survey we realized that a large percentage of these cases had deviated nasal septa, small nostrils, the presence of lymphoid tissue in the pharynx, particularly back of the posterior tonsillar pillars, —(cause or effect?) and a history of repeated tonsil and adenoid operations with poor results. Two cases had polypi (in one nostril only). A few cases had prominent vertebrae in the naso-pharynx.

In going over the diet we found that most of these cases had a positive dislike for milk and seldom drank any—the use of cream and butter was more or less limited in many cases. Some cases, however, used a well balanced diet.

In the symptomatology of these cases we found that the most frequent were nasal obstruction and nasal discharge. Very few complained of any pains, some had frequent nose bleeds; one complained of chronic bronchitis; but almost all were subject to frequent "head colds." Sneezing was present in but few of these cases.

*Read before the New York Academy of Medicine, Section on Laryngology and Rhinology, April 25, 1923.
*Accepted for publication in "The Laryngoscope", June 29, 1923.

Strange to say, most of the mothers of these cases stated that their children were active and acted as other children at play.

The following illustrates, perhaps, the data as presented by the average case—Kate S., aged 10½ years, has had nasal discharge since March, 1922, which is yellow, thick and considerable. She had an A. and T. operation in July, 1922, after which she showed some improvement for a short time, but now she is the same. She occasionally sneezes. Her appetite is poor; does not like milk but uses some cream and butter; bowels are apparently normal. This winter she had a slight chest cold; no history of joint or eye trouble. However she has had a constant head cold. This patient is very pale, rather small for her age and seems to be undernourished. Hair, teeth and skin are normal.

Nasal examination shows a spur on each side of septum, low down; considerable pus on floor of each nostril; some pus present in the naso-pharynx. X-ray showed mild involvement of ethmoids and antra.

All of our cases are being X-rayed before and during treatment. As yet no bacteriologic study has been made.

In the treatment of these cases we used simple hygienic measures; such as suction-irrigation by means of the Lore apparatus, followed by argyrol or bland oily solutions. All cases were advised to use cod-liver oil in addition to milk, cream and butter.

With such simple means we were able to get some improvements in some cases; but in all truthfulness we are loath to say that these good results are permanent. A fair number showed no appreciable result, which fact makes us believe that more energetic measures must be resorted to. As a result of the above experience we have outlined the following steps in the treatment of these stubborn cases.

1. Tonsil and adenoid operation when first operation was unsuccessful.
2. Correction of diet where indicated.
3. The use of cod-liver oil in all cases.
4. Nasal hygiene by means of suction-irrigation, argyrol lubricants, etc.
5. Antral lavage when indicated.
6. Correction of deflected septa when drainage is interfered with.
7. Uncapping of ethmoids and sphenoids only in severe cases when the above treatment has not helped, and in cases of polypi.

244 West 71 St.

OSTEOMYLITIS OF THE ORBIT FROM EXTENSIVE SINUS DISEASE.

Dr. James Albert Morgan, Honolulu, T. H.

C. L., Chinese, age 55; laborer, sugar plantation. Early history obscure. Came to Hawaii thirty years ago, during the days of the monarchy, as a contract laborer. About ten years ago the first symptoms appeared with a foul, purulent discharge from the left nostril, following an attack of fever. During the subsequent six months there was a gradual protrusion of the left eyeball, with no apparent impairment of vision. The purulent discharge continued during the next five years, being better and worse at times and the protrusion of the eyeball remained about the same. During this time the right nasal passage became involved, with protrusion of the right eyeball. During all this time the patient had been able to perform the duties of a plantation laborer, which meant long hours in the hot sun and hard manual labor. Two months ago the patient commenced to lose weight and became so weak that he could no longer continue his work. He had used Chinese herbs during this time but had never consulted a physician concerning his condition. Chinese fear a hospital and many times suffer the extreme rather than enter such an institution.

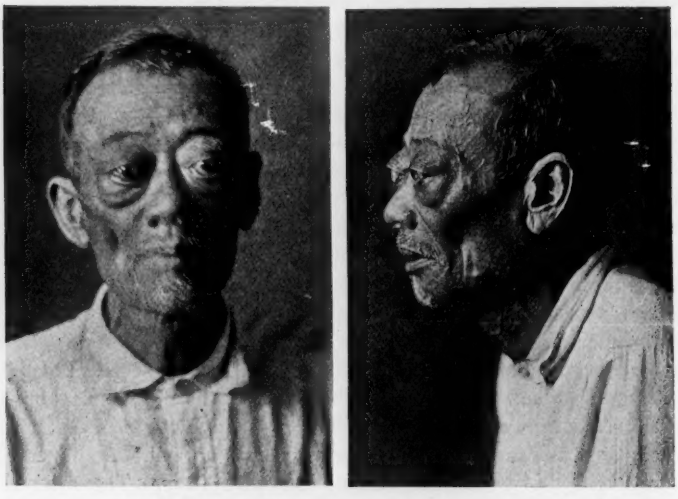
Eye examination showed a marked proptosis of both eyeballs, with only a slight impairment of the action of the extra-ocular muscles. Tension normal both eyes. Vision: both eyes .20/40.+Ophthalmological examination. Anterior media, normal. Both nerve heads and the surrounding retina somewhat hazy. The arteries were very tortuous and the veins overfilled. The color and form fields were normal.

X-ray of the nasal sinuses showed a general cloudiness extending into each orbit.

Nasal examination showed masses of polyps, large and small, studding the inferior and middle turbinates. From high up on the nasal septum two large polypoid masses, bathed in pus, hung down into the posterior naso-pharynx on either side. With such an extensive disease process, one is at a loss where to begin and where to stop. First, the maxillary sinuses were investigated and not only found filled with pus and polyps, but the entire nasal and alveolar walls were soft and necrotic. The nasal wall of each maxillary

*Accepted for publication in "The Laryngoscope", March 29, 1923.

sinus was removed almost en masse and the cavity cleared of its diseased contents. The anatomical relationship of the ethmoid, spenoid and frontal sinuses was not recognizable, but just a mass of soft, spongy tissue, covered with polyps and bathed in pus. The process also extended into the cribriform plate. The necrotic process was so universal that it was not possible to remove it all. The orbital walls were probed and found in a similar state of disease. The patient reacted well from the operation and there was practically no hemorrhage. During the week subsequent to the operations the proptosis slowly disappeared. General weakness continued however, and at the end of three weeks there was a sudden rise of temperature with bulging of both ear drums. Both tympanic mem-



branes were incised and sero-pus escaped. On the following day the patient became unconscious. The temperature ran a septic course and did not drop following the free drainage of the middle ears. The spinal fluid was under pressure and presented a high cell count. Death followed without the patient regaining consciousness. Autopsy showed a rarefying osteomylitis of both orbits with purulent lepto-meningitis.

This case is illustrative of the remarkable resistance of the tissues of the mucosa of the nasal accessory sinuses. This necrotic process had existed for so many years that the writer was reluctant in attempting surgical interference. Indeed it is this class of cases that too often place the rhinologist at his wits' end.

Honolulu, T. H.

THE DIRECTOSCOP OF HASLINGER (VIENNA) IN THE DIAGNOSIS AND SURGERY OF THE LARYNX.*

DR. SIDNEY ISRAEL, Houston, Texas.

There have been many advances in the field of laryngology during the past decade, but no one accomplishment occupies a more important place in the diagnosis and surgery of the larynx than that of Killian's method of suspension laryngoscopy. This procedure, while possessed of many advantages well known to all of us, like many others, also contains several disadvantages, especially as it applies to the amount of trauma occasionally taking place during the instrumentation in this type of direct laryngeal exposure. Possessing all of the advantages of the suspension laryngoscope,

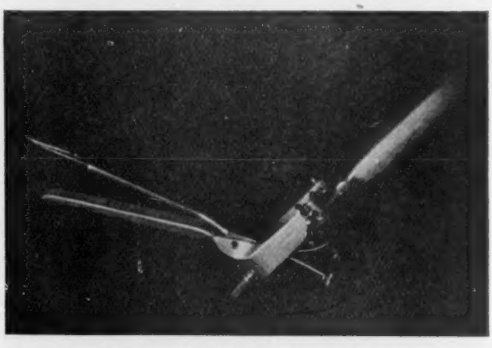


Fig. 1. Showing instrument with the tongue and pharyngeal spatula slightly separated. The hypo-pharyngeal plate is pushed forward slightly.

and in addition many distinct advantages of its own, and containing none of the objectionable features of any method, or instrument, for direct work within the larynx, up to the present time, is the Directoscop of Haslinger.

The Directoscop of Haslinger seems to be the ideal instrument for endoscopic exposure of the larynx. It is a self-retaining apparatus on the principle of a bi-valve speculum having two essential parts, to which are given the names, tongue spatula and pharynx spatula, and also a hypo-pharynx plate. To the pharynx spatula,

*From the Department of Laryngology, St. Joseph Hospital.
*Accepted for publication in "The Laryngoscope", Nov. 12, 1923.

at its distal end, is attached a movable metal plate for the hypo-pharynx; this plate is self-adjustable and controlled by means of two rods, which allow the plate to be pushed forward and pulled backward at the proper time to effect complete exposure. The metal plate is, by a spring connection, self-adjustable after being placed in position. After the instrument is properly introduced, the exposure of the larynx is controlled by means of a wing bolt, which causes the tongue spatula and pharynx spatula to be opened or closed at will, with either the right or left hand. The handle of

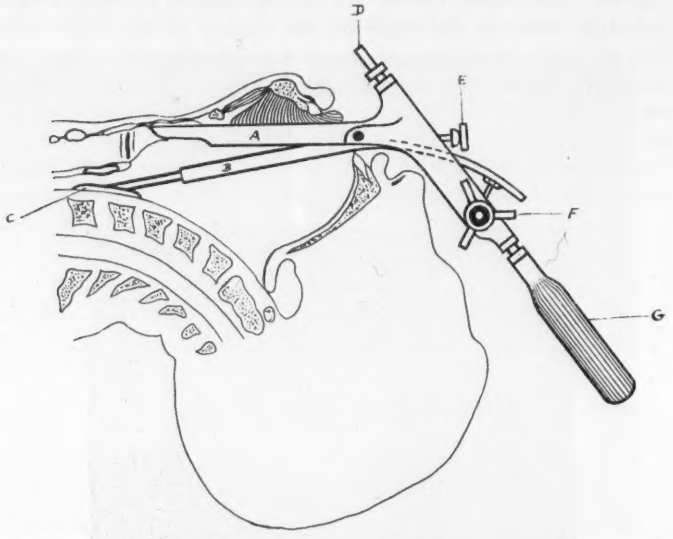


Fig. 2. Diagram showing instrument in place, in the recumbent position, and relationship of the hypo-pharynx plate to the cervical vertebrae. The distal end of the spatula holds the epiglottis, so that complete view of the larynx is obtained. A—Tongue spatula; B—Pharynx spatula; C—Hypo-pharynx plate; D—Metal post for attachment of handle or light; E—Metal rod for pushing forward or withdrawing hypo-pharynx plate; F—Wing bolt for controlling the spread or opening of the tongue and pharynx spatulae; G—Handle.

the instrument is adjustable to a metal post, either above or below, so that it can be conveniently manipulated, depending upon whether the patient is in an upright or recumbent position. The principle of this instrument for exposure of the larynx differs from other instruments heretofore utilized for this purpose, in that the hypo-pharynx and the underlying vertebrae are used as a fixed point from which counter pressure is produced to advance the tongue and epiglottis forward, thereby exposing the larynx.

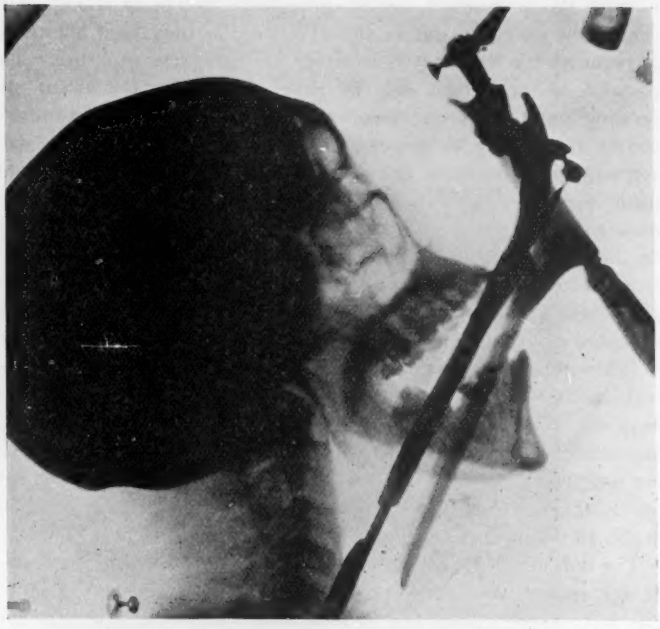


Fig. 3. X-ray photograph of the directoscop in place, in the sitting position, exposing the larynx completely.

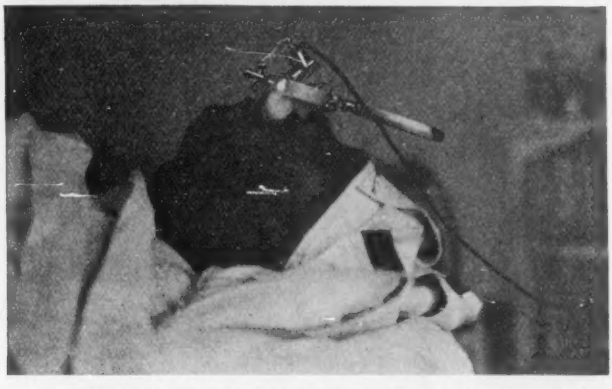


Fig. 4. Photograph of the instrument in situ, in the recumbent position, local anesthesia. You will note from this photograph that electric light attachment is utilized for the continuous illumination of the field of operation.

With this instrument, general anesthesia, except in young children, is the exception rather than the rule, as only local anesthesia is required for the most satisfactory and complete exposure. The position of the patient may be either upright or recumbent, depending upon the requirements. There is no gallows necessary, nor is it necessary to suspend the patient by the lower jaw, with ofttimes great pressure against the teeth. The exposure is complete, including the hypo-pharynx, the larynx, the anterior commissure, trachea and esophageal entrance. There is no pain, nor do the patients complain of post examinational discomfort. There is an entire absence of shock, and the instrument can be kept in place over a long period without the usual distress seen following exposure of this character.

When the instrument is in place, its position remains fixed and both hands are free to carry out any surgical or medical measure required. The usual dangers or mishaps we have been forced, heretofore, to be mindful of, in instruments of the suspension type, are not present in the exposure of the larynx with this instrument, nor is the time consumed in introduction nearly so long, due, no doubt, to the mechanical simplicity of the instrument and technic.

The indications for use of the Directoscop are the same as would be for suspension laryngoscopy. With the exposure afforded by this instrument, cysts have been dissected from the pharyngo-epiglottic fold, papillomas removed from the region of the anterior commissure beneath the vocal cords, cauterization of tuberculous lesions carried out, and a thorough examination of the entire larynx afforded under direct view, perfectly illuminated and exposed, with both hands free to execute any therapeutic measure necessary.

403-407 Second National Bank Bldg.

STRICTURE OF THE ESOPHAGUS FOLLOWING SCARLET FEVER.

Dr. Porter P. Vinson, Rochester, Minnesota.

Because of the apparent rarity of stricture of the esophagus following scarlet fever, I wish to record two cases that have come under my observation.

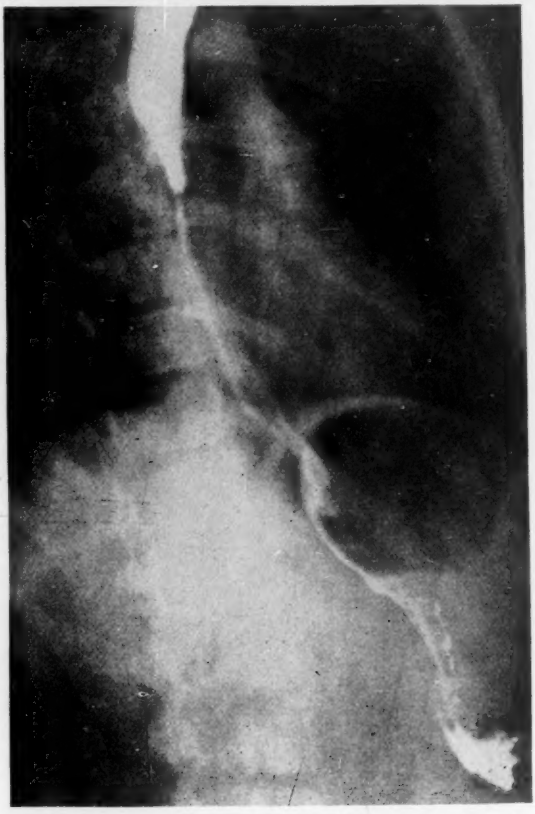


Fig. 1. (A367507). Benign stricture of the esophagus following scarlet fever in a child eight years of age.

REPORT OF CASES.

Case 1 (A367507), D. S., a girl eight years of age, was brought to the Mayo Clinic for examination, August 6, 1921, complaining of

*Accepted for publication in "The Laryngoscope", June 5, 1923.

difficulty in swallowing solid food. She had an attack of scarlet fever in November, 1920, complicated by bilateral suppurative otitis media, and one month after this illness she choked while eating an apple. Following this experience she had continued to have difficulty in swallowing solid food, and her diet had consisted

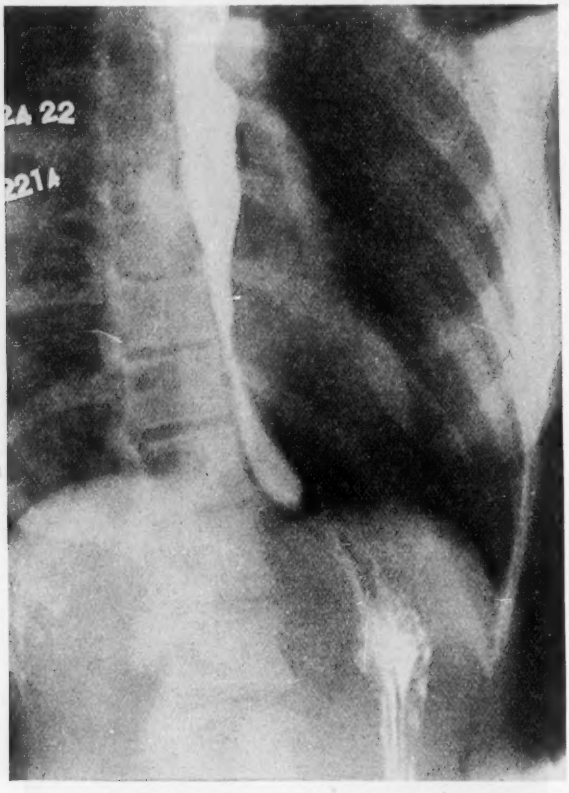


Fig. 2 (A392214). Stricture of the esophagus of thirty-one years' duration following scarlet fever.

chiefly of milk, eggs, and soft cereals. Whenever an attempt had been made to swallow solid food it was immediately regurgitated with a considerable amount of mucus. The patient was 7 pounds below her former weight.

A roentgenographic examination of the esophagus revealed an obstruction at the juncture of the middle and lower third of the or-

gan (Fig. 1). On passing a sound a stricture was located 25 cm. from the incisor teeth. This was easily dilated to 38 F., but the child was not able to remain under observation long enough to obtain permanent relief. However, in spite of insufficient dilation, her present condition is apparently quite good and she is able to swallow better than before the stricture was stretched.

Case 2 (A392214), Mrs. L. J. H., a woman aged thirty-eight years, came to the Clinic May 22, 1922. She had an attack of scarlet fever at the age of seven, and immediately afterward, difficulty in swallowing was noted. This would last only one or two days at a time, and occurred rather infrequently. There was relatively little discomfort from the age of ten to twenty-three years. At the age of twenty-eight years esophageal closure was complete for several days, and two years later a similar attack lasted for six days. During the latter part of February, 1922, there was another period of complete closure, and for two weeks before examination she had been able to swallow liquids only.

Roentgenographic examination revealed obstruction in the lower portion of the esophagus (Fig. 2), and on passing a sound, the stricture was located 33.8 cm. from the incisor teeth. The stricture was gradually dilated to 42 F. with complete relief from dysphagia.

It is not possible to determine whether the stricture in these cases was produced by the healing of an ulceration in the esophagus, or had followed the contraction of periesophageal scar tissue from a complicating mediastinitis, or mediastinal lymphadenitis.

REPORT OF A CASE SHOWING THE RESULTS OF THE HALLE AND THE LAUTENSCHLAGER OPERATIONS FOR OZENA.*

DR. WILLIAM MITHOEFER, Cincinnati.

I should like to present to you the report of a case showing the end-results of the newer operations for ozena.

Miss H., age 19, was first seen February 21, 1921. At this time she was complaining of an odor from the nose and nasal discharge, both of which had existed for at least seven years. Nasal examination showed the usual atrophic mucous membrane and offensive crusts of ozena. The turbinate bones especially the inferior, were greatly atrophied. Blood Wassermann—negative.

Conservative treatment was used for some time without relieving the patient of a severe supra-orbital headache. On January 31, 1922, an intra-nasal ethmo-frontal operation relieved the patient of the headaches but the crust formation and odor continued. On December 9, 1922, the Halle operation for ozena was done on the left side.

Technique of Halle operation—The inferior turbinate and that portion of the septum lying opposite are scarified, an incision is made on the lateral wall of the nose from the anterior end of the middle turbinate to the anterior end of the inferior turbinate. Another incision is made along the floor of the nose joining the incision at the anterior end of the inferior turbinate. The mucous membrane is now elevated along the floor of the nose and on the lateral wall underneath the inferior turbinate. A chisel is now placed in the incision on the lateral wall and an opening made into the antrum at the most anterior portion of the incision. After an opening is made the bone is further severed along the entire length of the naso-antral wall, being careful however, not to penetrate the bone completely in the region of the middle turbinate for fear of injuring the lachrymal apparatus. The chisel is now placed underneath the dissected mucous membrane on the floor of the nose and the wall of the antrum severed along the entire length corresponding to the naso-antral floor. A large sized periosteal elevator is now placed through the upper open-

*Read before Cincinnati Academy of Medicine, May 7, 1923.

ing made into the bone and the lateral wall displaced into the nasal cavity. When this is done a view of the antrum and ethmoid is easily obtained. The antrum if diseased, is then curetted and the ethmoid cells exentrated. The antrum is packed with iodoform gauze beginning above and packing as tight as possible. If reaction is not very severe the packing is allowed to remain six days. Dressings are changed every fourth or fifth day for at least a month. If the operation is properly done the inferior turbinate will be in close contact with the septum and a synechia will result. It is not advisable to divide the synechia as the nasal obstruction which is present immediately after the operation slowly disappears with the occurrence of a slight recession of the naso-antral wall.

The result of this operation in the patient I am presenting this evening, has been fairly satisfactory. Occasionally we find a few crusts in the region of the prelachrymal recess. It is our belief that the antrum which is usually very much involved in these patients, has not been sufficiently curetted through the intra-nasal opening.

On March 28, 1923, upon the suggestion of my associate, Dr. J. Maliniak, whose privilege it has been to observe personally the technique as employed by Lautenschlager, we decided to operate upon the right side according to this method. This operation is technically more difficult than the Halle operation. Our method of operating briefly told was as follows:

1. Incision under local anesthesia along the alveolar ridge, the incision a little longer than the one employed in the Denker operation and also placed a little lower on the alveolar ridge. 2. After careful dissection of soft parts and periosteum, two rhomboidal pieces of bone about one-half inch long were removed from the antral wall of the superior maxillary bone. These pieces of bone after being carefully shaped were placed aside for future transplants. 3. The antrum cavity was curetted and a large amount of hyperplastic tissue removed. 4. With a large periosteal elevator the naso-antral wall in the region of the middle meatus and ethmoid region was infracted toward the septum. 5. The ethmoid cells were next curetted through the antrum opening. The sphenoid cavity was diseased and was also dealt with. A small flap of nasal mucosa was placed into the antrum in the region of the natural orifice to insure good ventilation of the cavity. 6. The nasal mucous membrane on

*Accepted for publication in "The Laryngoscope", Sept. 25, 1923.

the floor and underneath the inferior turbinate was now carefully elevated beginning the dissection at the apertura pyriformis. One of the bone transplants taken from the facial wall is now placed over the floor, the other underneath the inferior turbinate on the lateral wall. Care was taken not to tear the mucous membrane for fear of infecting the bone transplants. 7. A flap of buccal mucous membrane with the base of the flap covering the apertura pyriformis was now placed in the antrum and the cavity packed with iodoform gauze. The first packing remained six days. The cavity was packed for four weeks when healthy epithelium had covered the antrum wall. The edges of the antrum cavity were then refreshened and the cavity closed.

This is in short the technique employed on the right side of the nose of the patient I am presenting this evening.

The practical significance of this operation is quite obvious. It removes radically all diseased tissue, it narrows the middle meatus by infracting the antral wall, it narrows the inferior meatus by the bone transplants and the antrum cavity is not closed until it assumes a healthy appearance. What was once a wide dry nose is now a narrow nose whose membrane instead of being atrophic is becoming slightly hypertrophic.

We have attempted the Lautenschlager technique on three other patients, the ultimate outcome of which will be discussed in a future series of cases when a more detailed description of the operation will be given. We consider the radical method of Lautenschlager to be more apt to give good results. We have modified the operation by not including Stenson's duct in the flap of buccal membrane as was originally described by Lautenschlager. The results in this case, six months after the first operation and two months after the second operation, were more than satisfactory. Crust formation has entirely ceased on the right side and is of negligible quantity on the left side. The odor has disappeared and the patient is highly pleased.

19 W. Seventh Street.

PHILADELPHIA LARYNGOLOGICAL SOCIETY.

May, 1923.

"Optic Neuritis, Secondary to Accessory Sinus Disease." Report of Case. Dr. I. B. High, (by invitation).

(*To be published in a subsequent issue of* THE LARYNGOSCOPE.)

DISCUSSION.

DR. GEORGE M. COATES: "This case, it seemed to me, to come under that class White speaks about as being the type of case where there is no marked evidence to account for atrophy but where we are justified in operating for the chances. White has, as Doctor High says, studied these cases closely and reached the conclusion that hyperplasis of the middle turbinate caused obstruction. In other words, there is interference with drainage and ventilation. One of the first cases I ever had was one sent to me with a diagnosis of early attachment of the retina. I could find nothing but that the retina looked a little suspicious. There was nothing in the case to suggest empyema, but having the courage of my convictions I opened it up and found pus. The patient recovered in a very few days. If we have hyperplasis it is our duty to operate upon the case and give proper drainage."

DR. S. D. WEEDER: "I had the good fortune to see a case which reminds me of this one in one respect. In examining the nose nothing had been seen. The case was then sent to me to see if there was a rhinological condition. I advised that an X-ray be taken by Dr. Pfahler. This was done and showed the ethmoid diseased. Dr. Posey and I did the operation and on reaching the posterior ethmoidal we found it was completely nocrosed; exactly like mush. The results of the operation were perfect."

DR. MACFARLAND: "I want to congratulate Doctor High on such an excellent paper. I had a case in which there was a total loss of nasal sign. The sudden onset he speaks of is quite characteristic. I find these cases regarded as transitory blindness. The correction of the septum may clear up cases. One of the medical students has a method of X-raying the ethmoid. He puts a plate in the nose to fit the sphenoid and takes the picture against that. It shows up the ethmoids excellently."

"Intra-nasal Carcinoma in Association with Nasal Polypi." Dr. Arthur J. Wagers.

DISCUSSION.

DR. P. S. STOUT: "I would like to mention a similar case I had some years ago. A woman who for years had been the head of an orphanage and had lived a very blameless life. She had always been a healthy woman and I found her family had been healthy, too. I was impressed with the fact, however, that one brother had a cleft palate, for which nothing had ever been done. In the fall of 1918 this woman saw a nose and throat specialist in this city, who said she had polypus. An operation was performed and she was relieved. The surgeon then went to Russia and the woman was referred to me. Owing to the fact that it looked a little different from polypus I had an X-ray taken. When the report came back I was shocked to find she had carcinoma. We used radium in rather heavy doses and she seemed very much better. I might add that after the first dose she almost died. That is not uncommon after the first treatment. Why, I do not know. The woman returned home and in about six months I saw her again. She was in a

deplorable condition. Her right eye was almost out of her head. We sent her to the hospital and removed the right eye, but a few months later she died. I could find nothing that might have caused her condition but three roots on the upper right side of her jaw."

DR. GEORGE M. COATES: "Dr. Wagers gives us an interesting case and asks questions that are extremely difficult to answer. As I remember the history she complained three years before. The chances are the woman had the disease. An over-looked irritation would seem to be predisposed to cancer. Two or three months ago one of the students at the hospital was taking out polypus from the nose of a colored woman. It looked very suspicious to me and I told him to send it to the laboratory. We received a report of carcinoma. It must have developed very quickly for they do not last a long time."

"The Indications for Mastoid Operations Under Local Anesthesia." Dr. P. S. Stout; Dr. W. G. Shemeley (by invitation).

To pe published in a subsequent issue of THE LARYNGOSCOPE.)

DR. W. G. SHEMELEY: "I feel perhaps I should apologize for venturing to discuss this paper since I have had but five cases under local anesthesia. They all occurred during my service in the army. Two were soldiers and three civilians. All had had influenza. They developed pneumonia, which was further complicated by empyema. Finally developed mastoiditis and one case had double mastoid abscess. Doctor Stout brings up the question of local anesthesia in mastoid operations. Some cases call for local anesthesia. I think many times it depends on the surgeon himself. Many of them prefer it. I think it makes for better technic, as the patient is awake and we are not apt to be careless and we make every move count. In conditions about the neck local anesthesia is many times preferable. In drug addicts I think I would use local anesthesia. In brain conditions again I would prefer general anesthesia. If the patient is fearful of local anesthesia better use general. I would prefer to overcome this fear with a little preliminary anesthesia."

DR. B. H. SHUSTER: "Doctor Ersner, if he were here, would tell you about cases we have done in the hospital. I have helped him in a number of them and they are usually done under local. We find that most of the patients object particularly to the chiseling. They do not like the sound and for that reason we do it quickly. As to our attitude in the operating room we find that by jollying the patient along we can do more with him."

DR. P. S. STOUT, (in closing): "If you have a lot of swelling it is rather difficult to give local anesthesia. But when it is a question of saving a life by giving a little pain, I think the patient would be willing to stand the pain. If I had a case of sub-periosteal abscess I would go ahead and use local in preference to ether.

"Intra-nasal Carcinoma in Association with Nasal Polypi." Dr. Arthur J. Wagers.

(*To be published in a subsequent issue of* THE LARYNGOSCOPE.)

"A Mastoid Bandage." Dr. Geo. M. Coates and Dr. Benj. H. Shuster.

(*To be published in a subsequent issue of* THE LARYNGOSCOPE.)

IMPROVEMENT OF DIPHTHERIA ANTITOXIN

NOTWITHSTANDING the vast fund of experience and information which has been gained through the many years in which Diphtheria Antitoxin has become established in medical practice, our knowledge is nevertheless steadily increasing and improvements continue to be made in the method of manufacture.

In the early days of serum therapy the standardization of antitoxin was a haphazard proposition, and even the tests utilized for safeguarding its purity left much to be desired. All that is a thing of the past. The standardization of antitoxin is now a definite and accurately controlled procedure, so that its potency, as expressed in antitoxic units, is a certain guide to the physician in determining dosage. Thoroughly dependable tests for insuring the freedom of the product from bacterial contamination or toxic substances of whatever nature have also been developed.

During recent years research effort has largely been directed toward increasing the concentration of antitoxin —getting the therapeutic dose in a smaller bulk and eliminating unnecessary solid material, especially proteins. An antitoxin thus refined has obvious advantages. The smaller quantity is easier for the physician to inject and less painful to the patient. Even more important, however, is the elimination of unnecessary albuminous substances which in certain patients may cause protein toxemia.

It is now possible, by methods of chemical precipitation, to so concentrate diphtheria antitoxin as to make a given volume many times as potent as the same amount of serum freshly separated from the blood of the treated horse. This is accomplished by precipitating the serum globulin, a constituent of the serum with which the antitoxic element is closely identified. Various methods of carrying out this concentration have been developed, the results of which vary—not only in the degree of the concentration, but also in the physical characteristics of the antitoxin thus obtained. It is very important that the concentration be effected without increasing the viscosity of the globulin to a degree sufficient to delay absorption when administered to the patient. Absorption is an important factor in the clinical response to antitoxin, whether the injections are given subcutaneously or intramuscularly; and delay in absorption is obviously a serious disadvantage.

The methods employed in the biological laboratory of Parke, Davis & Co. furnish a practical solution to this problem. The resultant antitoxin has a high concentration, a low content of protein in comparison with its unit strength, and on injection is absorbed with great rapidity.

To safeguard the potency of the product, every lot contains 40 per cent excess units at the date of issue—more than enough to insure the full labeled potency within the period of use for which it is dated.

Five sets of purity tests are carried out with every lot of antitoxin, to preclude the possibility of any contamination of the serum.

The development and rigid enforcement of these methods has given to the medical profession an antitoxin of high excellence, the purity and dependability of which are beyond all question.

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